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CHRONIC KERATOCONJUNCTIVITIS ASSOCIATED WITH NOCARDIA

WILLIAM L. BENEDICT, M.D.*

AND

HERMAN A. IVERSON, M.D.

Fellow in Ophthalmology, Mayo Foundation

ROCHESTER, MINN.

Reports on Nocardia, a subdivision of the genus Actinomyces, as a cause of disease of the eye are rarely found in the literature. A small number of cases of ocular infection due to Streptothrix, a synonym for Actinomyces, can be found. Various clinical observations have been made on streptotrichal infection of the eye. Superficial punctate keratitis,[1] yellowish nodules on the conjunctiva,[2] adherent yellowish white membrane on the palpebral conjunctiva[3] and raised nodules on the surface of the cornea or sclera[4] are a few of these. Although the usual infection lasts for years, 1 case was found in which streptotrichal infection of the eyes with adenitis and folliculosis lasted only three weeks.[5] Liégard and Landrieu[6] separated Nocardia from Streptothrix försteri as a cause of chronic conjunctivitis with mucopurulent secretion. Cavara[7] summarized data on fungous infections of the eye.

REPORT OF A CASE

The patient, a Saskatchewan woman 23 years old, was first seen at the Mayo Clinic in January 1940. The disease in the eye had begun soon after an attack of scarlet fever at the age of 7 years. Episodes of ocular pain, lacrimation, photophobia, dimness of vision and injection of the conjunctivas, lasting from one to several weeks, had occurred. Between attacks a state of relative comfort and improved visual acuity had prevailed. Slight entropion began at the age of 17 years. Treatment with copper sulfate and sulfanilamide had been without effect on the chronic inflammation.

Examination at the clinic revealed visual acuity of 3/60 in the right eye and 6/10 in the left. The right eye showed divergence of 10 degrees. Puckering of the eyelids, due principally to photophobia, was present (fig. 1 *A*). A few cilia were directed inward, and a gelatinous grayish white discharge was present. The inner surfaces of the eyelids presented either large patches of bright red granulation tissue or patches of scar tissue (fig. 1 *B* and *C*). No true follicles or papillae were present. With the biomicroscope small arterial trees and also superficial bullae were seen on the bulbar conjunctiva. Both minute and gross bands of scar tissue divided the surfaces. Posterior symblepharon was present. There was moderate injection of both bulbar conjunctival and scleral vessels. The entire right cornea was involved in interstitial keratitis. Finely granular infiltration was present throughout, and there was gross as well as microscopic vascularization of all parts of the cornea (fig. 1 *D*). The vessels were just below Bowman's membrane in the central portion of the cornea, but at the limbus they were at all levels. Several dirty white deposits were present between larger loops of vessels in the center of the cornea. Except for two small vascularized nebulas near the periphery, the left cornea appeared normal (fig. 1 *E*).

The patient's condition has remained nearly stationary during the four years since she first came to the clinic. The corneal lesions have progressed slowly, and she has had almost constant irritation, photophobia and discharge from the eyes. Administration of sulfanilamide, potassium iodide, zinc sulfate and metaphen have failed to help her. The sodium salt of penicillin, 1,000 Oxford units per cubic centimeter of solution, was applied by drop every hour for two weeks when the patient was not asleep, without noticeable effect.

BACTERIOLOGY

Eight cultures made at various intervals over a period of four years yielded, without fail, an organism which, according to the nomenclature

* Section on Ophthalmology, Mayo Clinic.

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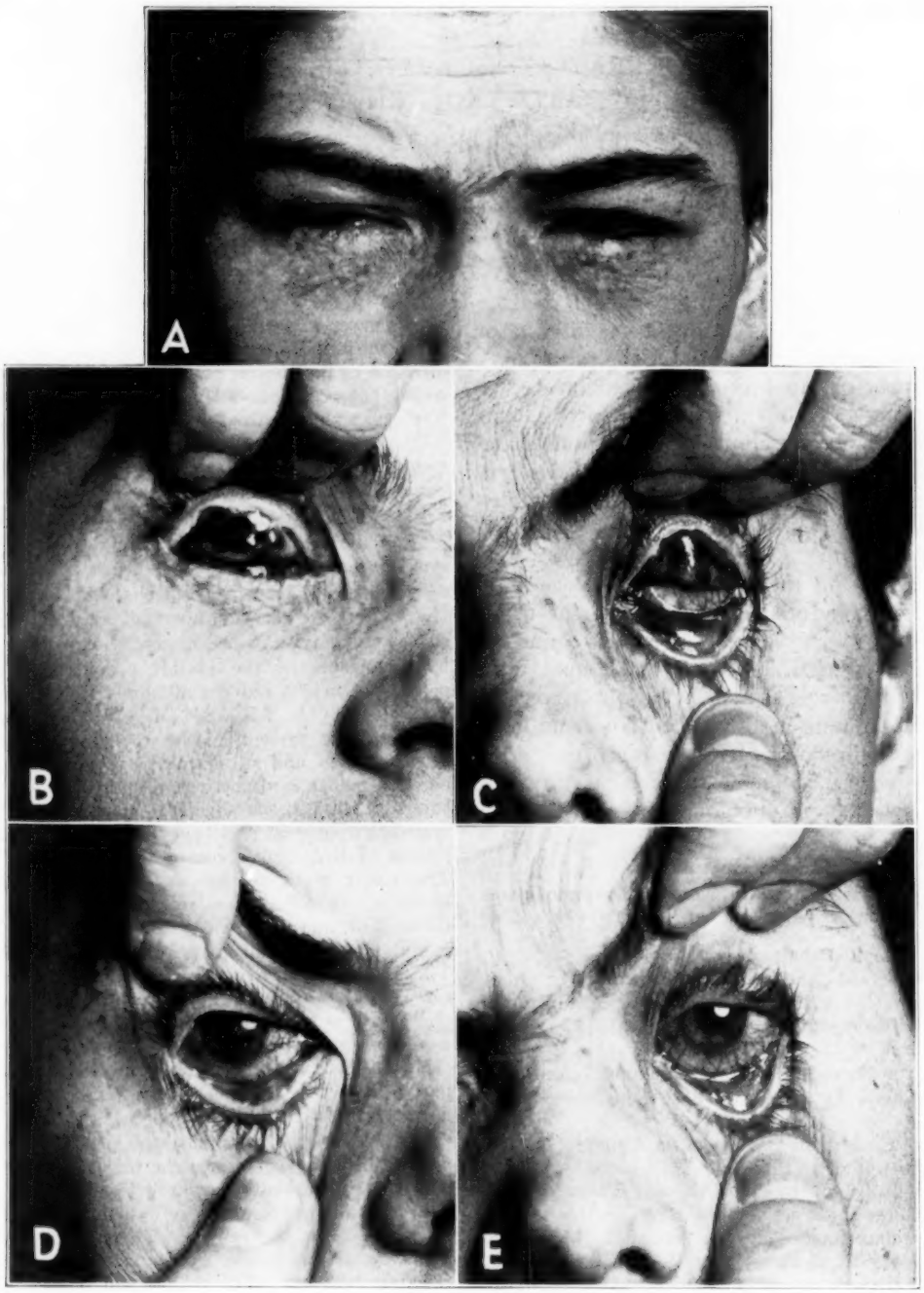

Fig. 1.—*A*, typical puckering of the eyelids. *B*, right eye, with large patches of granulation tissue under the upper lid. *C*, left eye, with regions of granulation tissue and scarring. *D*, right eye, with infiltration and vascularization of the cornea, with small white deposits in the center. *E*, left eye, with injection of bulbar conjunctival and scleral vessels.

recommended by Waksman and Henrici,[8] should be classified as a Nocardia. Other organisms persistently found were: alpha hemolytic streptococci, diphtheroid bacilli, micrococci and occasionally Staphylococcus aureus.

The Nocardia grew readily on all common laboratory mediums. On blood agar the surface colonies were roughly granular and had irregular edges (fig. 2*A*). The umbonate shape is easily seen in figure 2*A*. The original light yellow color turned to a deep yellow after several weeks. When it was placed in a tube of nutrient broth, a thin yellowish film was found at the surface within six hours. Shaking the tube broke up the film into small particles, which would not remain in suspension but gradually settled to the bottom of the tube. After incubation for forty-eight hours, the surface film had climbed 6 mm. up the sides of the tube. The surface glistened and appeared dry. After several days' growth, the film thickened and numerous deep yellow punctate accumulations appeared on the surface.

The organism, a gram-positive pleomorphic rod, was typically grouped in palisades in the fifteen hour broth culture (fig. 2*B*). A typical mycelium was formed when the organism was incubated in a drop of plain nutrient agar between a cover slip and a glass slide. No branching or spores were seen under varying conditions. The mycelium always broke up into single rods when transferred to a slide for examination. Galactose, dextrose, sucrose, levulose and maltose were fermented, with formation of acid but not of gas. Salicin, inulin, raffinose, xylose and

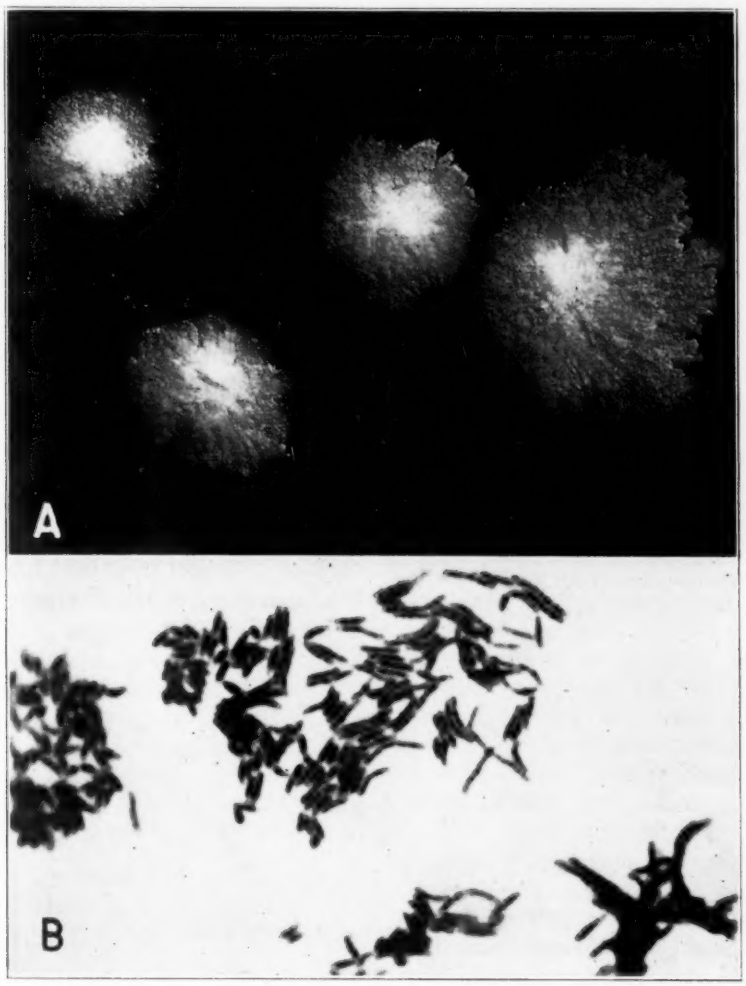

Fig. 2.—*A*, colonies of Nocardia on blood agar incubated at 37 C. for three days (× 6). *B*, Nocardia organisms from a fifteen hour nutrient broth culture. The organisms are gram-positive (× 2,000).

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lactose were not fermented. Gelatin stab cultures revealed good surface growth but no liquefaction after two weeks at 37 C. The catalase reaction was positive. The methyl red reaction was negative. Nitrates were converted to nitrites, with formation of the ammonium ion. The organism grew only between a p_H of 5.5 and 9.5. On litmus milk there was surface growth but no visible change in the medium. Indole was not converted to indigotin. There was no growth under anaerobic conditions. Yellowish punctate colonies were produced on washed potato. There was no coagulation of fresh human serum in four days and no liquefaction of coagulated horse serum in one week. It did not grow on Czapek's sucrose medium or with paraffin in Czapek's solution in one month. The organisms were not acid-fast when stained by the Ziehl-Neelsen method but were partially acid-fast to Umbreit's [9] modification of the stain. The sodium salt of penicillin diluted with nutrient broth to a concentration of 1 Oxford unit per cubic centimeter completely inhibited growth of the organism.

EXPERIMENTATION ON ANIMALS

Subcutaneous injection of living Nocardia organisms into a guinea pig, intravenous, intracerebral and intraperitoneal injections into mice and intravenous and intraperitoneal injections into rabbits caused no morbidity. Necropsy of the guinea pig after one and a half months revealed nothing abnormal. Necropsy one week after intraperitoneal injection of the organisms into mice revealed a few white nodules in the spleens, pancreases and mesenteries. Microscopic sections of the lung of a rabbit after intravenous injection revealed a nonspecific foreign body reaction.

Living organisms were injected at several different times into the corneas and conjunctivas of a rabbit. Control injections of heat-killed organisms were made. There was little difference in the foreign body reaction caused by the living and by the killed organisms. Gradual absorption of the injected material was the rule.

COMMENT AND SUMMARY

Various unclassified types of Actinomyces are present in soil. The possibility of these organisms getting into the eyes is certainly not remote. Bakly stated that streptotrichal organisms are found on normal conjunctivas. It appears that the pathogenicity of this group of organisms is low and also that only a small percentage of persons are susceptible to this type of infection. When infection is once established, treatment is usually unsuccessful.

In this case the chronic keratoconjunctivitis was due, most likely, to a Nocardia. The bacteriologic studies contained herein were done in the laboratory of Dr. Fordyce R. Heilman.

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SARCOIDOSIS WITH RETINAL INVOLVEMENT

REPORT OF TWO CASES

MAJOR S. GOLDBERG* AND LIEUTENANT F. W. NEWELL†
MEDICAL CORPS, ARMY OF THE UNITED STATES

Boeck's sarcoid was described by Longcope and Pierson [1] as "a chronic infectious granuloma, persisting often for years, sometimes spreading slowly from one organ or tissue of the body to another, frequently relapsing, seldom producing serious constitutional symptoms, resistant to treatment, but at times healing spontaneously." The most frequent site of ocular involvement is the iris. Blegvad [2] stated that iritis occurs in 10 per cent of the cases. Osterberg,[3] however, found but 27 cases of iritis in 500 cases of Boeck's sarcoid in the literature and estimated that involvement of the iris occurs in more nearly 5 per cent of the cases. The lacrimal gland and the conjunctiva are involved infrequently. The most uncommon sites of sarcoid infiltration are the choroid and the retina. King [4] stated that sarcoid must be considered as an extremely rare cause of localized chorioretinitis, an assertion which is definitely borne out by the scarcity of reported cases.

The earliest report of chorioretinitis accompanying Boeck's sarcoid is that of Hudelo and Rabut,[5] who described in 1925 a patient with the disease who presented iridocyclitis and bilateral choroiditis *"de nature bacillaire."* Reis and Rothfeld [6] in 1931 described a 17 year old white girl presenting cutaneous sarcoid of the Darier-Roussy type and roentgenographic evidence of osteitis cystica tuberculosa (Jüngling) and of enlargement of the hilus glands. Exophthalmos and papilledema were present in both eyes, together with secondary atrophy of the optic nerve in the right eye. The left eye presented a white tumor mass, elevated 20 diopters, with an uneven surface covered by retinal blood vessels. Vision was reduced to counting fingers at 40 cm. with the right eye, and the left eye was blind. The patient died in an epileptiform attack, and necropsy revealed sarcoid involvement of the brain, the optic nerve and the left retina, with generalized tuberculosis elsewhere.

Horton, Lincoln and Pinner [7] described a white woman, aged 35, with recurrent uveitis in whom sarcoid changes were found in enlarged supraclavicular nodes removed for histologic study. The right eye showed evidence of previous iritis, and there were multiple inflammatory lesions in the lower half of the choroid with involvement of the overlying retina suggestive of tuberculosis. An exudate present in the pupillary area of the left eye prevented view of the fundus. Meyer [8] observed a patient, aged 24, with recurrent iritis and secondary glaucoma of the right eye and periphlebitis of the left inferior nasal vein with sarcoid present elsewhere.

Walsh [9] reported a white woman, aged 34, who showed blurring of the temporal margin of the optic nerve in the right eye, with slight pallor and numerous superficial, discrete white spots in the paramacular area, with a tendency to perivascular arrangement. The left eye showed white spots scattered throughout the fundus. One year later a mass, elevated 8 diopters, was present in the left eye and considered to be tuberculous. Nine years later the left eye was phthisic and was enucleated; sarcoid was demonstrated microscopically in the retina.

One of the 7 patients with Boeck's sarcoid reported by King [4] showed an active area of chorioretinitis in the left eye, which was healed two years later. King also examined an eyeball sent

* Chief of Eye, Ear, Nose and Throat Section, LaGarde General Hospital.

† Assistant chief of Eye, Ear, Nose and Throat Section, 108th General Hospital.

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to him for microscopic study and found typical sarcoid changes in the retina and in the distal portion of the optic nerve.

Because of the scarcity of reported cases we believe it is of interest to report the following 2 cases which were observed during the past year at LaGarde General Hospital, New Orleans.

CASE 1.—A 23 year old Negro, single, was first seen Sept. 4, 1942, complaining of painful swelling of the face and nodules in the inguinal region. He first noted nodular swelling in the right inguinal region June 25, 1942, which was followed by a similar swelling on the opposite side. About Aug. 20, 1942, the parotid glands became moderately enlarged, and the posterior cervical, axillary, subclavicular and epitrochlear nodes became swollen. A marked diminution of vision in the right eye was noted at this time.

The family history was not relevant. There was no record of tuberculosis or venereal disease.

Physical examination revealed a moderately well nourished Negro, not acutely ill. The parotid glands and the lacrimal glands were somewhat enlarged and not tender. There was generalized, painless lymphadenopathy, with the nodes enlarged to the size of a lima bean. Neurologic examination revealed no abnormalities.

Vision in the right eye was reduced to perception of movements of the hands, and in the left eye was 20/20. The pupils were equal and reacted normally to light and in accommodation. Tension was 26 and 30 mm. of mercury (McLean) in the right and the left eye respectively. Slit lamp examination showed no evidence of inflammation of the iris. There were much pigment and many blood cells in the anterior portion of the right vitreous. The right fundus showed numerous deep and superficial hemorrhages with thrombosis of the superior temporal vein, which had been replaced by a fibrous band. The arterioles were distinctly attenuated, and a severe edema was present in the macular area. The left fundus was normal.

Roentgenographic studies of the chest showed adenopathy of the hilar nodes of both lungs and a generalized increase of markings throughout both lung fields. No abnormalities of the bony structures of the hands or the feet could be demonstrated.

The blood showed a persistent hypochromic anemia, with the red cell count varying from 3,450,000 to 4,440,000 per cubic millimeter and the hemoglobin content varying from 6.3 to 7.4 Gm. per hundred cubic centimeters. The white cell count varied from 3,450 to 8,750 per cubic millimeter, with eosinophils from 8 to 27 per cent. Cultures of blood and results of inoculation of guinea pigs with sputum were repeatedly negative. Repeated examinations of sputum revealed no acid-fast organisms. The Kahn and the Wassermann reactions were negative. Serum protein was 6.3 Gm. per hundred cubic centimeters, with 2.55 Gm. of albumin and 3.75 Gm. of globulin. The sedimentation rate varied from 44 to 67 mm. per hour. Agglutination tests with Brucella melitensis and Pasteurella tularensis gave negative results as did a cutaneous test with Frei antigen. The Mantoux reaction was negative with 1 mg. of old tuberculin.

Repeated biopsies were made of enlarged nodes, and their microscopic appearances were similar. The normal structure of the lymph nodes was almost completely destroyed and replaced with numerous small tubercles. These were composed of epithelioid cells without a zone of lymphoid tissue, with giant cells of the epithelioid type present. In a few tubercles under high power a small central portion of necrosis was seen. Boeck's sarcoid was the diagnosis of the pathologist (Major Sloan Wilson), and it was subsequently confirmed by the Army Histopathological Center.

Six weeks after the patient's admission a white mass, extending 3 diopters into the vitreous, was present at the inferior, nasal margin of the disk, and partially overlying it. The mass was superficial to the retinal blood vessels, of irregular surface, with many new blood vessels overlying it, and with sharply demarcated margins. The mass remained constant in size, and the vitreous cleared considerably in succeeding weeks, with all of the hemorrhages disappearing without sequela. A mass of new blood vessels extended from the disk forward into the vitreous, and the inferior nasal vessels were surrounded by whitish areas of retinal and choroidal destruction.

Early in the course of the disease the patient had a temperature never exceeding 100 F., which subsequently did not recur. The swelling of the parotid glands disappeared, the lymphadenopathy became much less severe and many nodes resolved spontaneously.

CASE 2.—The patient, a 24 year old Negro soldier, married, was first seen Jan. 27, 1943, complaining of swelling of the face and nodules in the arms and the forearms, which had been increasing in number the previous three months. He stated that after a hypodermic injection in September 1942 he noted "knotlike" nodules in the left forearm. In December 1942 swelling of the parotid glands developed, and he was hospitalized at a station hospital with the diagnosis of mumps. The swelling did not subside, and the patient was transferred to LaGarde Hospital.

The family history waas not relevant and contained no record of tuberculosis. With the exception of pneumonia at the age of 12, the patient had had no previous illness.

Physical examination revealed a well nourished Negro, not acutely ill. The parotid glands were symmetrically enlarged, with nodules palpable immediately under the skin, and the indurated glands were felt beneath these. There was very slight tenderness to palpation. Deep in the subcutaneous tissue of the upper, outer angle of the orbits nodules the size of a pea were palpated. The posterior cervical, subclavicular, epitrochlear and inguinal glands were slightly enlarged and felt like small, hard nodules. The arms and the forearms contained subcutaneous nodules, varying in size from that of a pea to that of an almond. The epithelium of the entire body was normal. Neurologic examination showed slight atrophy of the deltoid and the biceps muscles, with weakness in the left arm.

Vision in the right eye was 20/50, not improved with correction, and in the left eye 20/20. The pupils were equal and reacted normally to light and in accommodation. Tension was 20 and 24 mm. of mercury (McLean) in the right and the left eye respectively. Slit lamp examination showed no evidence of involvement of the anterior segment. Ophthalmoscopic examination showed the right disk to be obscured in the inferior nasal quadrant by a grayish white mass approximately 2 disk diameters in size and extending nasalward. The mass was overlying the retinal blood vessels and projected 4 diopters into the vitreous. Numerous new blood vessels were present over the mass, which had sharp margins and was apparently encapsulated. Inferior and superior to the mass sub-

hyaloid hemorrhages were present. Small flame-shaped hemorrhages in the periphery and one adjacent to the macula were seen. The veins were of irregular caliber and constricted; the arterioles appeared normal.

Roentgenographic studies of the lungs revealed only an increase in the hilar markings. No pathologic condition of the phalanges could be demonstrated.

The white cell count varied between 5,300 and 10,950, with a normal differential count. Eosinophils never exceeded 3 per cubic millimeter. The red blood cell count varied between 4,600,000 and 5,600,000, with a hemoglobin content of 90 per cent. Urinalysis revealed no abnormality. The Wassermann and the Kahn reaction were negative on repeated examinations. The serum protein was 7.8 mg. per hundred cubic centimeters, with 4 mg. of albumin and 3.8 mg. of globulin. The sedimentation rate was 79 mm. per hour.

A cutaneous test for tuberculosis gave a negative reaction with 1 mg. of old tuberculin. Agglutination tests with P. tularensis and B. melitensis gave negative results, as did a cutaneous test with Frei antigen.

Biopsy specimens were taken of a parotid gland, the right lacrimal gland and a subcutaneous nodule in the forearm. The report was essentially the same for each. The entire tissue was infiltrated with small lymphocytes and plasma cells, and there were numerous small tubercles present. These had an extremely narrow outer zone composed of fibrous tissue and lymphocytes, with a central area composed largely of fat cells, through which coursed small capillaries, with a few epithelial cells present. Each tubercle was moderately infiltrated with polymorphonuclear leukocytes, with no epithelial giant cells or foreign body giant cells present. There was a decided increase of connective tissue throughout the whole tissue. The histologic picture was such that it most nearly conformed with that of a sarcoid of the Darier-Roussy type.

The infiltration of each tubercle by polymorphonuclear leukocytes made the absolute diagnosis of sarcoid difficult. However, Colonel D. E. Ash of the Army Medical Center stated: "The histologic picture is distinctly atypical for an out and out diagnosis of sarcoid of the Darier-Roussy type, which, however, we favor against other possibilities." Microscopic sections of the biopsy specimens have been submitted to dermatopathologists throughout the country for opinions.

Five months after the onset of the swelling of the parotid glands the patient began to have almost daily slight hemoptysis, accompanied by gastrointestinal symptoms. Inoculation of sputum into a guinea pig gave negative results on two occasions. Repeated examinations of sputum and gastric washings did not reveal the presence of the tubercle bacillus. No pathologic condition of the intestinal tract could be demonstrated by roentgenograms, and studies of the stools revealed normal conditions. The swelling has remained constant, but there has been a slight decrease in the size of the subcutaneous nodules.

Vision in the right eye has improved to 20/30 without correction, and all hemorrhages have disappeared without residue. The mass overlying the disk has not increased in size, and new vascularization has apparently stopped.

The 2 cases reported have many of the clinical signs of sarcoid and are similar to each other in regard to the enlargement of the parotid and lacrimal glands, the numerous nodules over the forearms and the appearance of the fundi. The initial diagnosis in the second case was made ophthalmoscopically, and we believe that possibly the lesions of the fundi in these cases are specific for sarcoidosis. The presence of these masses in the retina indicates the possibility that sarcoid may cause either an exudative ocular inflammation, as characterized by iritis, with or without an accompanying chorioretinitis, or a proliferative lesion, as occurred in these 2 cases. The masses observed in these cases probably represent a true sarcoid infiltration of the retinal tissue, as occurred in the cases of Reis and Rothfeld [6] and King,[4] in which the eyes were studied microscopically.

The retinal vascular changes are much more difficult to explain but may simply represent mechanical interference with the blood supply. However, in Meyer's [8] case the periphlebitis was a striking part of the ophthalmoscopic picture, and it may be, therefore, that the vascular changes are an essential part of the disease early in its course. In the first case here reported the vascular changes preceded the visible tumor mass, and possibly this sequence occurs in all cases of infiltration of sarcoid tubercles in the retina. Nothing specific for the disease was noted in the appearance of the fundi at this stage.

The infiltration of polymorphonuclear leukocytes in the nodules removed for microscopic study in case 2 is distinctly atypical and has made the absolute diagnosis of sarcoid difficult. However, clinically the case presents many of the symptoms and signs of sarcoid, and we believe that it represents a true instance of this disease.

Several considerations were essential before a diagnosis of sarcoidosis was made. Coats's disease, characterized by a massive exudation, with or without hemorrhages, occurring particularly in early adult life, bears some resemblance to the clinical picture visualized. In Coats's disease there is little or no generalized disorder. Cholesterol crystals are sometimes present.

Tuberculosis of the choroid of such extensive degree that a mass is present is practically always accompanied by anterior uveal involvement. Reactions to tuberculin were negative; negative reactions to tuberculin are the rule in sarcoidosis. Acute exudate choroiditis of more than moderate degree is usually accompanied by sufficient change in the aqueous to produce keratic precipitates. White blood cells in the vitreous are numerous. A blood dyscrasia or lymphosarcoma was a possible diagnosis, but each was eliminated by findings in the lymph nodes and by studies of the blood.

The condition in the second patient had been previously diagnosed as von Recklinghausen's disease, before he was admitted to LaGarde Gen-

eral Hospital. We did not see the very early stages, but at the time of admission the mass appeared to be overlying the retina, partially obscuring the disk. New vessels were seen passing over the edges, and anteriorly, as if the mass were encapsulated. The presence of widely spread nodules was suggestive of neurofibromatosis. However, clinical studies revealed evidence of sarcoidosis.

To recapitulate, in each of these cases whitish irregular masses, extending far forward into the vitreous, were present. Periphlebitis and hemorrhages were a prominent feature. In case 1 hemorrhage was extensive, both preretinally and in the retinal layers. There were absence of keratic precipitates, an optically clear aqueous and very little inflammatory response in the vitreous. The involved eye was always white. The formation of new blood vessels was striking early in the course of the disease, suggesting that the lesion would progress to a retinitis proliferans but with eventual absorption of the mass in each case. The retina remained in place. The end picture in each fundus was similar. There remained discrete whitish areas not unlike those seen after an exudative choroiditis.

No treatment was attempted in either case. Mydriatics were employed only for inspection.

SUMMARY AND CONCLUSION

Two cases of sarcoidosis with involvement of the retina are presented, the first an instance of the Boeck type and the second of an atypical Darier-Roussy sarcoid. It is believed that the lesions of the fundi described may be specific for a proliferative type of sarcoid infiltration of the retina.

WHITE RINGS OF THE CORNEA

REPORT OF MICROSCOPIC EXAMINATION

M. W. JACOBY, M.D., AND RAFAEL DOMINGUEZ, M.D.
CLEVELAND

Only a small number of cases have been reported so far of the characteristic opacity of the cornea first described by Coats.[1] To the 42 cases collected by Bietti,[2] including 4 of his own, 2 cases of MacRae [3] and 5 of Waldman [4] may now be added.

In 46 of the 50 cases available, only one eye was affected and only one ring was present. One of Ballantyne's [5] patients had two rings in one cornea; 1 of Kuan's [6] had one ring in each cornea; 1 of MacRae's [7] had one ring in one cornea and several rings in the other cornea, and 1 of Coats's [8] had multiple rings in both eyes.

The rings occur in any part of the corneal field. By slit lamp examination the white deposit is seen in or adjacent to Bowman's membrane.

The rings cause no symptoms. As a matter of curiosity we should like to recall that 1 of the patients of Kuan [6] saw the white spot in the mirror and came to the clinic to have the "foreign body" removed.

The lesions are stationary for long periods. Waldman [4] did not notice any change in the lesions in 2 cases which he was able to observe for five and seven years respectively.

An idea of the rarity of the condition is given by Kuan,[6] who in two years found 11 cases of the disease among 8,032 patients coming to the clinic for the first time. In an ophthalmologic practice extending over twenty years, one of us (M. W. J.) has encountered only 5 cases.

Nothing is known concerning the cause of this lesion. Coats [1] expressed the opinion that it is congenital but brought no facts in support of this idea. Lampert,[9] Gallemaerts [10] and others stated the belief that the rings are produced by trauma. However, the frequency of the rings is exceptional when compared with the frequency of trauma to the eye (as pointed out by Bietti [2]), and in only 1 case has the ring been found within a nebula.[3] Ballantyne [5] and others remarked on the frequency with which the white rings are associated with either a previous history or the actual presence of some intraocular disease. Yet not 1 of the 11 patients of Kuan [6] had evidence of intraocular disease.

With regard to the pathogenesis of this lesion, two hypotheses have been advanced. Ballantyne,[5] after mentioning the drusen of Bowman's membrane described by Rindfleisch [11] and Elschnig,[12] suggested that the white rings could arise "from degenerative change, calcareous or otherwise, in such drusen." Kuan [6] stated the belief that the lesion is due primarily to a disease of the terminal branches of the corneal nerves and that this hypothetic disease may lead to a necrosis of Bowman's membrane, either by the action of some poison or by trophic disturbance. Kuan speculated further that the ring results from a secondary change—proteic precipitation, fatty degeneration or mineral incrustation—in the periphery of the necrosed part of Bowman's membrane.

From the Department of Ophthalmology and the Department of Pathology, St. Luke's Hospital.

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9. Lampert, cited by Gallemaerts.[10] Of the cases cited by Gallemaerts, Ballantyne [5] retains only those of Lampert.

10. Gallemaerts, E.: Examen microscopique des affections de la cornée au moyen de la lampe à fente, Paris, Masson & Cie, 1926.

11. Rindfleisch: Arch. f. Ophth. **37**:236, 1891; cited by Ballantyne.[5]

12. Elschnig, A.: Drüsenbildung an der Bowman-'schen Membran, Klin. Monatsbl. f. Augenh. **37**:453, 1899; cited by Ballantyne.[5]

The structure of the lesion is not well known. Only three authors have reported on the microscopic examination of excised rings.

Muramatsu,[13] according to Uyama,[14] found a homogeneous purple-staining substance under Bowman's membrane, between this membrane and the substantia propria of the cornea. The epithelium, although thin, was intact and level over the opacity.

Uyama [14] removed the white ring of his patient with a needle and after fixing the piece of cornea with a solution of formaldehyde stained

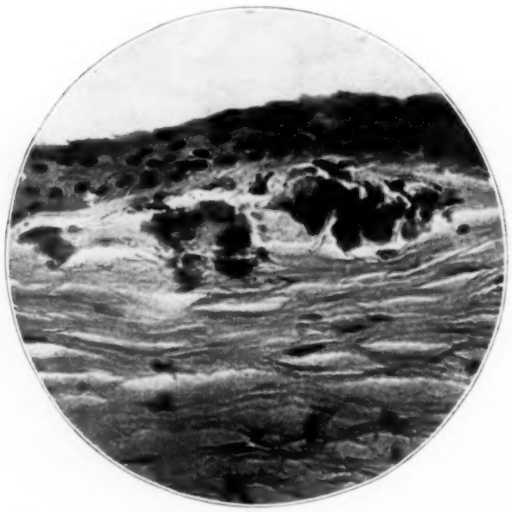


Fig. 1. — Photomicrograph of a section of cornea (hematoxylin and eosin; × 320) showing the part of the ring farthest from the limbus.

it with sudan III. With the microscope he saw a small mass of orange-yellow granules in the superficial layer of the cornea. Uyama concluded that the white rings are certainly composed of fat. Since a nuclear stain was not used, he could not determine the relation of the granules either to the epithelium or to Bowman's membrane.

Bietti [2] took biopsy specimens of the rings of 3 of his 4 patients. The specimens were fixed in a solution of formaldehyde, embedded in gelatin, sectioned and examined for fat. In only 1 of the 3 biopsy specimens was there a lipoid infiltration of Bowman's membrane, but this infiltration was rather diffuse and by no means confined to the zone containing the white ring. In the other 2 specimens, the result of the examination was rather meager. The only abnormalities mentioned consisted of a thinning of Bowman's membrane at some points, a lack of homogeneity of this membrane at some other points and, in 1 specimen, local disappearance of Bowman's membrane. Below Bowman's membrane Bietti noticed a few newly formed cellular elements, which he interpreted as connective tissue elements, and also a few masses of an amorphous degenerated tissue resembling hyalin.

We have had the opportunity of examining one of these rings after enucleation of the eye for some other condition. In view of the incomplete knowledge of both the structure and the chemical composition of this rare opacity, we think the report of our examination is of interest.

REPORT OF A CASE

A 55 year old Italian laborer came to the ophthalmic clinic May 1, 1936, with the complaint of poor vision.

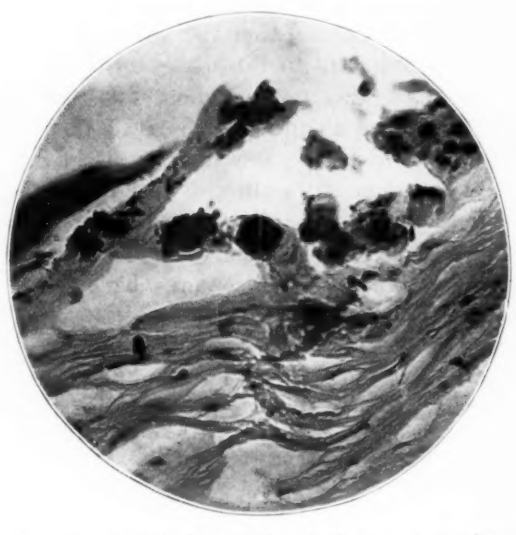


Fig. 2. — Photomicrograph of the same section of cornea as in figure 1 showing the part of the ring nearest the limbus. The tear of the tissue is undoubtedly due to the brittleness of the cluster.

Vision was 6/30 in the right eye and 3/60 in the left eye. Examination disclosed immature cataracts, principally of a posterior saucer shape type, and hyperrefringence of the nucleus. No abnormalities were noticed in either fundus, but the details of the left were poorly seen.

External findings were unimportant with the exception of a white ring, 1 mm. in diameter, located in the lower nasal quadrant of the right cornea near the limbus. With the corneal microscope the ring was seen to consist of conglomerate chalky dots located at the level of Bowman's membrane. Within the ring

13. Muramatsu, N.: Five Cases of White Ring in Cornea, Chuo-Ganka-Iho **27**:32, 1935; cited by Uyama.[14]

14. Uyama, Y.: White Ring in Cornea, Arch. Ophth. **15**:309 (Feb.) 1936.

there were many discrete dots of the same character. The overlying epithelium was apparently normal, and the remainder of the cornea was also normal. There were no indications of previous disorder of the eye and no history of injury to the eye.

The results of a general physical examination were essentially normal. The upper right molar teeth presented periapical absorption and were extracted. A Wassermann test of the blood gave a negative result, but the reaction to a Kline test was positive. Since no history and no signs of active syphilis were present, no antisyphilitic treatment was advised at this time.

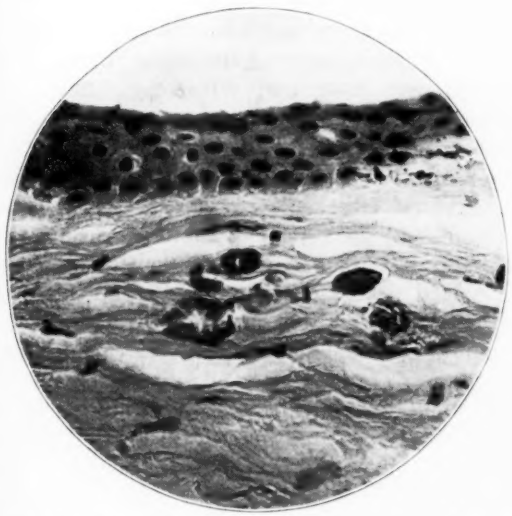


Fig. 3.—Photomicrograph of the same section of the cornea as in figures 1 and 2 showing the part of the cornea inside the ring.

On Nov. 2, 1936, a combined extraction was performed on the left eye, with no operative or postoperative complications. The ophthalmoscopic examination of this eye showed a clear vitreous and a normal fundus. Vision with correction was 6/6.

On June 17, 1938, a combined extraction was performed on the right eye, with no operative or postoperative complications. The ophthalmoscopic examination of the fundus disclosed no abnormalities. The vision in this eye was not corrected to better than 6/12.

On Jan. 6, 1939, vision of the right eye was reduced to 6/24, the eye was quiet and a marked hypotony existed. A fairly clear view of the fundus showed no retinal detachment or evidence of a pathologic condition. An extremely thin capsular membrane was present but, owing to the hypotony, discission was not performed. The left eye was quiet and normal.

From the time of the first admission to the early part of 1939 all of seven Kline tests on the patient's blood gave positive results. The Wassermann test made at the same time gave positive results in three of the seven tests. In April 1939 the result of an examination of the spinal fluid was negative. The patient was considered to have latent syphilis and was treated rather lightly from April to October 1939. (The patient's wife has been treated intensively for syphilis since 1929.)

On Feb. 2, 1940, the patient reported to the clinic, stating that the right eye had been painful for several weeks. Vision was reduced to light perception, and there was distinct ciliary injection. The tension (Schiøtz) was 33 mm. of mercury. The iris was bulging forward, up to the posterior surface of the cornea, and obliterating the anterior chamber. The capsular membrane was also bulging forward, and a view of the fundus could not be obtained. The left eye was quiet, and the corneal microscope showed no evidence of inflammation of the eye.

Enucleation of the right eye was advised, and the eye was removed on Feb. 27, 1940.

On the last examination, June 10, 1942, the serologic reactions of both blood and spinal fluid were negative.

Microscopic Examination.—Immediately after enucleation, the cornea was excised and was fixed in dehydrated alcohol. After fixation, the part of the cornea containing the ring was embedded in paraffin and sectioned.

In the preparations stained with hematoxylin and eosin three clusters of bluish globules were seen. Two of these clusters lay just underneath the epithelium

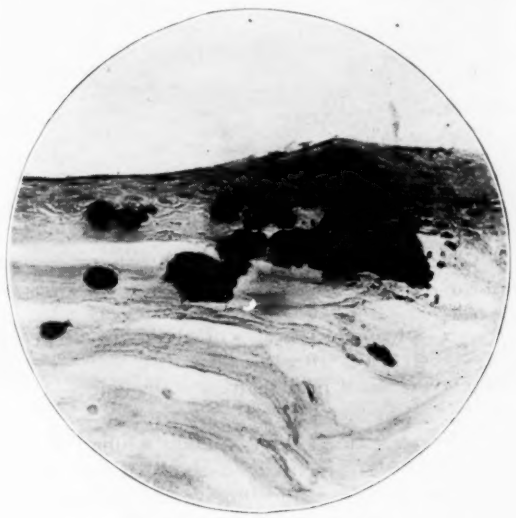


Fig. 4.—Photomicrograph of a section of cornea stained by Kossa's method (× 320). This is the only section of this cluster that did not tear.

(figs. 1 and 2) and a third between the other two but deeper in the substance of the cornea (fig. 3). The first two photomicrographs represent sections of the ring itself, and the third represents a section inside the ring. The globules were either circular or ovoid, and when ovoid the long axis was usually parallel to the surface of the cornea. The largest globule measured 31 microns in longest diameter and 19 microns in smallest diameter. The deepest surface of the deepest globule lay 64 microns below Bowman's membrane. A few of the globules could be located exactly in Bowman's membrane. (See, for instance, the globules

farthest to the left in figure 1.) Others, especially those within the ring, lay clearly below Bowman's membrane and showed no connection with this membrane (fig. 3). Still a few others were present above Bowman's membrane, actually within the epithelium (fig. 4). The majority, however, lay in or adjacent to Bowman's membrane, corroborating the results of slit lamp examination. No hyaline masses (drusen) were seen above Bowman's membrane either inside or outside the ring. In the ring proper, where the deposit was heaviest, Bowman's membrane could not be seen, even after decalcification. Figure 4 shows an elevation of the corneal surface on top of the cluster, but, since the cornea was not fixed in situ, this demonstration does not prove that the cornea was deformed before enucleation.

The material of the globules is not homogeneous, and in some a definite crystalline arrangement can be seen. After treatment with a dilute solution of acetic acid the globules practically disappeared. In sections treated by Kossa's method the globules became deep black (fig. 4).

Fig. 5. — Photomicrograph of the same section as figures 1, 2 and 3 after decalcification and restaining (showing the same field reproduced in figure 1).

The section from which the first three photomicrographs were made was subsequently treated with potassium ferrocyanide and hydrochloride acid as a test for iron. The globules were almost dissolved out without the formation of prussian blue. The section was then restained with hematoxylin and eosin. This time the clusters appeared as indistinct masses of a faintly stained amorphous material. Directly above the remnants of the cluster reproduced in figure 1, there were found several elongated nuclei, which stained with hematoxylin fully as deeply as the nuclei of the overlying epithelium but more deeply than the nuclei of the corneal corpuscles (fig. 5). By comparing figure 5 with figure 1, one can see that these nuclei were visible in the original cluster before decalcification, but we could not identify them. In all probability these filaments represented the deformed nuclei either of connective tissue cells, as suggested by Bietti, or of wandering cells immobilized in the neighborhood of the calcific deposits. No other foreign cellular elements were seen, and no vascularization of the cornea was found.

The sections of the globe after fixation in solution of formaldehyde and embedding in paraffin showed the usual appearance of secondary glaucoma without inflammation of the vascular membrane.

COMMENT

From the foregoing examination it appears that the deposits forming the white rings contain calcium phosphate and no iron. Other substances are undoubtedly present because the clusters do not disappear completely under the action of acids. Our examination does not allow us to assert either the presence or the absence of lipids, but the report of Bietti [2] indicates that lipids are not a constant constituent of the rings. In regard to the examination for calcium it is well to remember that practically all fixatives—except dehydrated alcohol—remove calcium from the tissues. Indeed, it is possible that by using a solution of formaldehyde as a fixative Bietti may have removed a great deal of the calcium present in his biopsy specimens. Otherwise it is difficult to reconcile the sharpness of the deposits seen both in vivo and in vitro (figs. 2 and 3) with the vagueness of Bietti's findings. But it is not difficult to reconcile the latter with the appearance of one of our decalcified clusters (fig. 5).

Our results lend no support to the speculative views of either Ballantyne [5] or Kuan [6] concerning the pathogenesis of the deposit. In areas of heavy calcareous deposit, Bowman's membrane is of course disorganized or even absent; but there are no drusen either outside or within the ring and there is calcification below an otherwise normal Bowman's membrane inside the ring (fig. 3). The distribution of the clusters does not suggest a nervous or pore distribution. Moreover, the preservation of Bowman's membrane inside the ring makes untenable the supposition of Kuan, that the deposits are laid down on the periphery of an altered segment of Bowman's membrane. In order to account for the shape of the lesion in accordance with Kuan's view, it becomes necessary to say that the degenerative disease of the nerves involves only the nerves in the periphery of a small circular area, a very unlikely assumption.

Saint Luke's Hospital.

INTRANASAL DRAINAGE FOR CURE OF CHRONIC INFECTION OF THE TEAR SAC

INITIAL, TRANSCANALICULAR, INVERTED U-SHAPED INCISION TO FACILITATE FULL OPENING OF THE TEAR SAC

DAVID J. MORGENSTERN, M.D.
Assistant Attending in Otolaryngology at the New York Post-Graduate Medical School and Hospital
BROOKLYN

Chronic tearing of the eye is usually due to blockage of the tear-conducting passages. The cause of the obstruction may be a scar due to injury, chronic inflammation of the mucous membranes leading to stenosis, a foreign body or pressure from a tumor. Most often chronic inflammation of the mucous membranes causes the obstruction, which is usually located at the bottom of the tear sac or in its extension downward into the nasal cavity, namely, the nasolacrimal duct.

Such interference with tear conduction predisposes to chronic infection in the blocked duct. To drain and cure this infection and to restore normal conduction of tears into the nose, a new opening is made above the obstruction through the bone that separates the tear sac from the nasal cavity. In making this opening from the nose to the tear sac, the greatest difficulty, as Bookwalter [1] stated, is encountered in enlarging the opening into the tear sac to the same size as the bony opening. There are two reasons for this: First, the sac is not easily visualized because it is located at the extreme end of the newly formed bony opening; second, the sac recedes from the instrument that attempts to seize it and is pushed farther out of the operator's sight because it is soft tissue, without firm backing for counterpressure when approached intranasally.

As an aid in visualizing the sac and as a means of securing counterpressure, a heavy Bowman probe passed through the canaliculus has been used to push the sac into the newly formed bony opening. This maneuver is of little avail when the sac is small, when it is softened by disease so that the probe perforates it, when a wide space separates the sac from the nasal cavity or when the sac is thickened or tied down by scar tissue. Such unusual scarring and thickening of the tear sac may result from severe inflammation or from laceration, fracture or previous operation in or about this area.

If the sac is thickened or filled with granulations, West [2] advised its intranasal extirpation. To accomplish this, extensive intranasal resection of tissues, namely, submucous resection of the nasal septum, middle turbinectomy and wide resection of the lateral nasal wall, are often necessary.

In this area, so difficult to visualize intranasally, I employ the keener sense of touch to replace vision.[3] By placing the forefinger over the tear sac, one can feel the bony margin surrounding it, as well as an instrument inserted into it, either through the canaliculus or through the nasal cavity. To insert an instrument, a probe, passed through the canaliculus into the tear sac, is first pushed through the bone into the nasal cavity. The end of this probe, projecting into the nose, guides increasingly larger operating hooks, which are slid along it through the bone into the tear sac. With the sense of touch as guide, the tear sac is fully opened. Electrocoagulation is used to destroy the torn portion of the sac and to coagulate the surface of the bony opening. This combats infection and helps prevent closure. This technic is adequate to provide drainage and to effect cure in the usual case of infection of the tear sac, whether purulent or catarrhal.

On the other hand, the opening of a scarred and thickened tear sac has hitherto proved most difficult. I now fully open such tear sacs easily by an added, initial step in technic. During the past year, I had the opportunity of operating in 2 cases of this unusual condition. In each instance the tear sac was so thickened and scarred that I had difficulty in opening it adequately.

Read by invitation before the New York Society for Clinical Ophthalmology, Jan. 3, 1944.

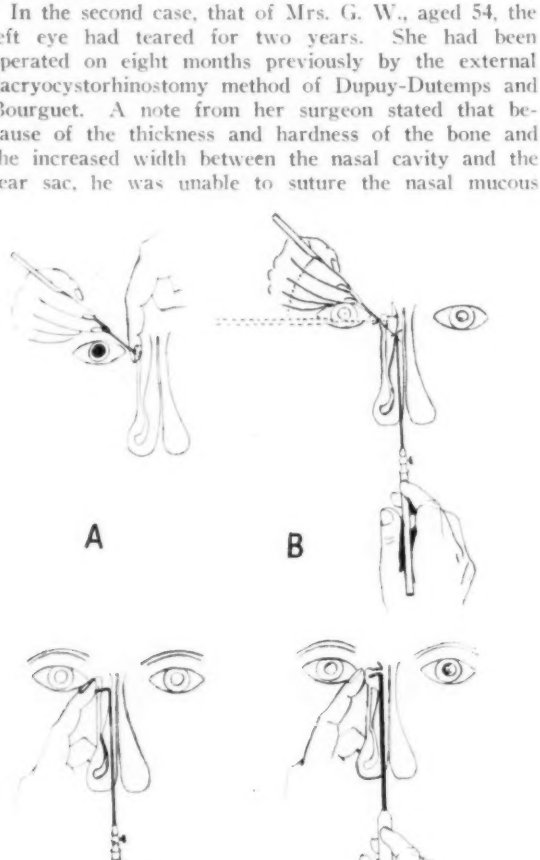
1. Bookwalter, C. F.: Intranasal Dacryocystostomy, Arch. Ophth. **49**:568, 1920.

2. West, J. M.: Die Technik der Eröffnung des Tränensackes von der Nase aus nach Erfahrung an 130 einschlägigen Operationen, Arch. f. Laryng. u. Rhin. **27**:504, 1913.

3. Morgenstern, D. J.: Intranasal Drainage for Cure of Chronic Tear Sac Infection: New Technic Aided by Electrocoagulation So Simplified as to Be an Office Procedure, Arch. Ophth. **27**:733 (April) 1942.

Mrs. R. B., aged 67 had a chronic infection of the tear sac of fifteen years' duration with frequent, severe acute exacerbations. I observed her during the last attack, which eventuated in abscess of the sac. After the pus had been evacuated externally and the opening into the sac had healed, I operated and encountered a much enlarged and thickened sac and a hardened lacrimal bone.

In the second case, that of Mrs. G. W., aged 54, the left eye had teared for two years. She had been operated on eight months previously by the external dacryocystorhinostomy method of Dupuy-Dutemps and Bourguet. A note from her surgeon stated that because of the thickness and hardness of the bone and the increased width between the nasal cavity and the tear sac, he was unable to suture the nasal mucous membrane to the membrane of the sac through the bony fistula formed. On operating in this case, I encountered hard bone and a greatly thickened and scarred tear sac.



A, initial transcanalicular inverted U-shaped incision to open the tear sac fully. *B*, small hook on a guiding probe penetrating the lateral nasal wall. *C*, lateral view of the sac from without, showing the posterior and the anterior (palpated) crest of the fossa sacci lacrimalis and movement of the palpated tip of the hook in the fully opened sac. The hook was the largest used. *D*, electrocoagulation of sac and fistula, guided by palpation.

In both cases I used sharper-pointed instruments to tear through and more and stronger electric current to coagulate the wall of the sac. In addition, a Bowmann probe, insulated except at its tip, was passed through the canaliculus to electrocoagulate further the wall of the sac. To give greater counterpressure, against which the point of the sharper instrument could cut through the toughened sac wall, the thumb nail was substituted for the softer palpating forefinger at the inner canthus of the eye. The tactile sensation of the thumb nail is adequate to guide safely the tip of the sharp hooked instrument in opening the sac. To secure greater concentration of the electrocoagulating current, the patient's metal electrode, usually placed in contact with the back, is rolled up and held in the operator's moistened palm, the index finger of which is firmly applied over the tear sac. This more strenuous procedure secured drainage and cure in these 2 cases of chronic infection of the lacrimal sac and restored normal conduction of tears into the nose.

As a result of the experience gained in these 2 cases, I adopted the additional, initial step in technic to facilitate full opening of the tear sac. I now begin by fully opening the tear sac, instead of leaving this most difficult step for the end. This enables me to take advantage of the bony wall of the lacrimal fossa, against which a thin knife, which has been passed through the dilated canaliculus, incises the wall of the sac, under the guidance of the palpating forefinger placed over the sac.

The tip of the knife is shifted to the most inferior part of the posterior lacrimal crest. From this point, the knife directed vertically upward, and always in firm contact with the bone, an incision is made upward to the top of the fossa along the posterior crest. Here, without its losing contact with the bone, the knife is turned at a right angle, and the top of the sac is cut through. Finally, with the edge of the knife turned downward through another right angle, the wall of the sac is cut downward as far as possible, about 1 mm. posterior to the anterior lacrimal crest. To assure complete continuity of the U-shaped incision, I apply electrocoagulating current to the knife, insulated except at its tip, while it incises the wall of the sac. Thus, an inverted U-shaped incision is made in the medial and in part of the posterior wall of the sac. The knife is then withdrawn. Since the top of the sac cannot be palpated, it is better to cut through it at a slightly lower level. Preliminary irrigation of the sac, through its being filled, aids in determination of the margins of the sac.

The top and bottom of the knife handle are notched along a line cut by a plane passing through the flat surface of the blade. The fingers feeling these notches correctly direct the knife blade while making the inverted U-shaped incision. The knife edge is so sharpened as to avoid cutting diametrically opposite surfaces of the canaliculus. Slight pressure is exerted on the dull side while the knife is inserted into the canaliculus. Local infiltration of tissues with the anesthetic should not be too great, for it

obtunds the sharpness of the lacrimal crests, which are the essential landmarks.

From this point, the procedure is practically the same as that described in my original paper.[3] A guiding probe is passed through the lower canaliculus into the bottom of the tear sac and is pushed into the nasal cavity at an angle of about 45 degrees. If several attempts to pass the probe at points posteriorly placed meet with resistance, a few sharp taps on the probe will usually perforate the sclerotic bone.

Examination of the nasal cavity now reveals the end of the guiding probe projecting into it. Four or five hooked instruments are fashioned from various sizes of stiff steel wire, the ends bent at 90 degrees and sharpened. The smallest of the hooks is slid along the back of the guiding probe, penetrating the lateral nasal wall. Palpating with the forefinger of the free hand at the inner canthus, the operator feels for movement of the point of the largest hook, employed last. The guiding probe is now removed.[4] Under guidance of the sense of touch, the bony lacrimal fossa is broken down as widely as possible. This is easily accomplished everywhere except anteriorly, where there is the hard bone of the frontal process of the superior maxilla. A sharp-toothed hook is used to rasp some of this bone. To avoid the strenuous rasping of all this hard bone, the anterior vertical incision through the sac is made about 1 mm. posterior to the anterior lacrimal crest. By removal of the bone underlying the medial and part of the posterior wall of the sac and by virtue of the initial, inverted U-shaped incision, a flap is formed. Irrigation with copious amounts of saline solution turns the flap down into the newly formed bony opening into the nose and washes out loose debris.

4. If the tip of the middle turbinate is enlarged, the obstructing portion is then excised.

Last, to combat closure, as well as to reduce infection, a large, dull-pointed hook, which has been insulated except at its bent end, is used in electrocoagulation of the newly formed opening into the infected tear sac, as well as the sac itself. At the bottom, where the flap has been turned down, the electric current is applied lightly in order to prevent the destruction of the flap. Occasional irrigations of the sac during the healing process maintain the position of the flap in the bony opening and overcome any tendency to stricture which may result from possible injury to the canaliculus. This flap helps to safeguard the permanence of the bottom of the bony opening and aids in epithelization of the bony fistula.

When bone for counterpressure is lacking, only the anterior leg of the inverted U-shaped incision can be made against the hard bone of the frontal process of the superior maxilla. Then the top and the posterior portion of the incision must be attempted with uncertainty. This situation is found in cases in which a previous operation has been performed by whatever method and in which an inadequately opened tear sac has closed while the opening through the bone remains. The same situation exists from time to time when there is a dehiscence or an exceptionally thin lacrimal bone, so that the knife easily perforates it.

One cannot be certain before operation whether the tear sac will prove to be scarred or thickened. Since the inverted U-shaped incision is easily and accurately made as an initial step, and not after the bony backing of the sac has been removed, I have adopted it as the initial step in all cases.

Since there is a minimum of tissue destruction and usually scarcely any bleeding, this procedure can be carried out in the office, the patient being able to leave after a short rest.

433 Eastern Parkway.

BUCCAL MUCOUS MEMBRANE GRAFTS IN TREATMENT OF BURNS OF THE EYE

RALPH SIEGEL, M.D.
WELCH, W. VA.

A review of the literature reveals a lack of emphasis on the urgency of immediate transplantation of buccal mucous membrane in the treatment of conjunctival and corneoconjunctival burns. Denig,[1] in 1912, first described the use of mucous membrane in severe lime burns, ammonia burns, and a burn caused by the contents of a golf ball. O'Connor,[2] in a brief review (1933), and Thies,[3] in a German monograph (1938), supported Denig. Von Grolman,[4] in 1940, in South America, reported an isolated case of a labial mucosa graft for a corneoconjunctival burn due to sodium hydroxide. The idea has not received the popularity in this country that it merits, however. Weeks,[5] in 1936, referred to the employment of buccal mucous membrane grafts for trichiasis, symblepharon and trachoma and in treatment of essential shrinkage of the conjunctiva in pemphigus, but he did not mention their use for burns of the eye.

According to Thies, burns of the eye present three stages: first, simple inflammation, in which the subjective complaints are similar to those of traumatic conjunctivitis; second, formation of a profuse exudate, and third, necrosis. Caustic alkalis and certain war gases, notably mustard gas (dichloroethylsulfide) produce corneal damage which, in spite of medical treatment, becomes progressively worse. In cases of such burns the severity of the injury may not be apparent at the time of the accident, but the lesion continues to grow worse. Sollmann[6] pointed out that alkalis penetrate the tissues, producing necrosis and soluble albuminates, the action of which continues for several days.

Villard and Delord[7] showed that when sulfuric acid was dropped into the eyes of rabbits the following changes took place: (1) death of the cellular elements of the cornea, with swelling of the corneal lamellas; (2) invasion of the cornea, the anterior chamber and the iris by leukocytes; (3) corneal perforation due to destruction of the cornea by the acid, and (4) cicatrization, in which the corneal opening may be closed by the protruding iris and epithelium from the adjacent conjunctiva.

In severe burns the significant pathologic feature is the necrosis of tissue and the destruction of the perilimbal circulation, including the lymphatics and the marginal plexus formed by the anastomotic arcades of the anterior ciliary arteries. A graft primarily restores the circulation to the limbus and thereby reestablishes the nutrition of the cornea and maintains the normal metabolism. The graft secondarily serves to replace destroyed conjunctiva, with prevention of the sequelae of symblepharon and entropion. Since living conjunctiva is not available in sufficient quantities, the next best substitute is living human tissue similar to conjunctiva itself. Buccal mucous membrane fulfils this crying need and produces practically no abnormal tissue reaction.

Other types of tissue have been used as material for transplantation. Wiener[8] did experimental work on rabbits and monkeys with preserved conjunctival grafts. Conjunctiva was dissected from cows' eyes, stretched over slabs of cork and placed in 4 per cent concentration of solution of formaldehyde U. S. P. Before being used, it was washed with tap water and saline solution. A complete symblepharon was produced in monkeys with concentrated lye, and a conjunctival graft was placed over molding material and secured with lid sutures. The steamy cornea cleared up in about two weeks. In a second monkey a piece of preserved conjunctiva 20 per cent larger than the area of the prepared surface

From the Department of Ophthalmology, Cook County Hospital.

1. Denig, R.: Am. J. Ophth. **3**:256, 1920; Circumcorneal Transplantation of Buccal Mucous Membrane as Curative Measure in Diseases of Eye, Arch. Ophth. **1**:351 (March) 1929.

2. O'Connor, G. B.: Early Grafting in Burns of Eye, Arch. Ophth. **9**:48 (Jan.) 1933.

3. Thies, O.: Die Verätzungen des Auges, Stuttgart, Ferdinand Enke, 1938.

4. von Grolman, G.: Arch. de oftal. de Buenos Aires **15**:429, 1940.

5. Weeks, W.: New York State J. Med. **36**:1372, 1936.

6. Sollmann, T.: Manual of Pharmacology, Philadelphia, W. B. Saunders Company, 1917, pp. 125, 131 and 137.

7. Villard and Delord: Arch. d'opht. **24**:515, 1904.

8. Wiener, M.: J. Michigan M. Soc. **42**:53, 1943.

was used to cover the defect, and no dental stent was employed. The graft was anchored solely by sutures to the conjunctiva of the host. The healing in this animal was even more rapid than that in the first monkey. The author emphasized that monkeys have more regenerative power than man. Since preservation destroys the life of the cells in the graft, this method depends solely on the ingrowth of cells from the surrounding tissues of the host. Wiener visualized the use of preserved heterogenous grafts in ophthalmic surgery when mucous membrane is needed.

De Rötth [9] recommended the use of fetal membranes in cases of symblepharon when buccal mucous membrane is not obtainable. He used this material in 6 cases of symblepharon and in 2 cases in which the socket had to be enlarged. The fetal membranes were obtained by cesarean section and were kept in tepid Locke's solution from one to fifteen hours. The author admitted that the late results were poor, with nearly total shrinkage of the membrane.

Clay and Baird [10] used grafts from the prepuce and labia minora for the correction of symblepharon and after the removal of large growths from the conjunctiva. These grafts are conspicuous, have a foul odor and desquamate.

Brown [11] advocated the use of a sheet of rabbit peritoneum as a protective covering to separate the cornea and the palpebral conjunctiva until healing is well advanced. The use of rabbit peritoneum as a covering does not stimulate vascularization in cases in which there is impaired perilimbal circulation. Furthermore, it may add insult to injury by exercising a deleterious effect on the cornea.

Zenkina,[12] in the recent Russian literature, tried transplantation of conjunctiva from cadavers for burns of the eye. The conjunctiva was taken from persons who had died of trauma, not later than eighteen hours after death. The material was kept at a temperature of approximately 2 to 4 C. in isotonic solution of three chlorides U. S. P. with 2 per cent dextrose for periods ranging from twenty-four hours to four days. Ten cases were reported. The author concluded that this method offers good results in stimulating the regenerative forces in the surviving tissue and that from the cosmetic angle it is preferable to the autoplastic buccal mucous membrane graft.

It is my purpose in this paper to call attention by means of case reports to the necessity for the prompt use of buccal mucous membrane in the therapy of conjunctival and corneoconjunctival burns. In this series of 7 cases of ocular burn at the Cook County Hospital, the burn was due to lye (a mixture of sodium hydroxide and sodium carbonate) in 5 cases, to lime (calcium oxide) in 1 case and to gasoline in 1 case. Data on an eighth case of burn due to sulfuric acid were furnished by the late Dr. Sanford R. Gifford and Dr. C. E. Jaeckle, of Chicago. These cases are not only of ophthalmic importance but of forensic interest, since in the 5 cases of burns with lye homicidal intent or intent to disfigure the features of a suitor was involved.

In the order of their severity, the agents causing ocular burns in this series of cases were: (1) lye, (2) gasoline, (3) sulfuric acid and (4) lime.

Buccal mucous membrane was obtained with ease and from the patient himself. The wound in the mouth healed promptly and was nonobjectionable. There was adequate buccal mucous membrane to supply tissue for the most extensive binocular burn, without too great a sacrifice to the patient's mouth. Except for a secondary buccal hemorrhage in 1 case, which was easily controlled, there were no complications from this angle. Although buccal mucous membrane retained its rosy color and thickness, anatomic and functional restitution was good.

Buccal mucous membrane grafts took in 6 out of 7 eyes which were grafted with this material. The graft sloughed in a case of lye burn of the left eye (case 1) which was so severe and extensive that the entire perilimbal circulation was totally and irreversibly destroyed. In this case the process went on to complete necrosis and corneal perforation in spite of a mucous membrane graft made ten hours after injury. This case indicates that when the circulation has been completely wiped out, there is little hope of salvaging the eye. Later, the subsequent contracted socket was reconstructed by a plastic procedure. The right eye in the same case suffered only a slightly less severe burn. Vision was reduced to counting fingers at 2 feet (60 cm.). There was severe necrosis of the palpebral and bulbar conjunctiva, with the typical "porcelain" appearance of the inferior half of the bulb, a sign indicating destruction of the perilimbal circula-

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tion in this area. A direct burn involved the lower three fourths of the cornea. With prompt grafting a good functional and cosmetic result was obtained. Vision in the right eye returned to 4/10 after three and a half months. The cornea cleared except for a hazy rim at the limbus from 5:30 to 9:30 o'clock, and the cul-de-sac healed, with no sequelae (figure, *A*). Three of the 4 eyes which received buccal mucous membrane grafts not later than ten hours after injury healed with functional and cosmetic results superior to those in eyes which were grafted later than ten hours after the burn.

In this series there were 2 cases of burn due to lye in which grafts were not made. In case 6, that of a bilateral burn with lye with direct corneal involvement and intact perilimbal circulation, equivocal local perilimbal vascular interference appeared in the left eye on the fifth day after the injury. This case occurring early in our experience, a graft was considered unnecessary, and the operation was not done. The cornea of the left eye showed delayed progressive corneal infiltration and a corneal ulcer, which appeared three weeks after injury. There was indolent healing, with the development of a large corneal leukoma of the left eye. Vision in the left eye returned to 4/10 only after a long, stormy convalescence.

A lye burn of the left eye, with direct corneal involvement and intact perilimbal circulation, was present in case 7. Since the perilimbal circulation was intact, a mucous membrane graft was not made. A large opaque central opacity developed in the left cornea, with poor functional return. Six weeks after injury vision in the left eye was correctable only to 1/10. It is interesting that infected corneal infiltrates at the periphery appeared ten days after the injury and that a timely delimiting keratotomy, as advised by Dr. Sanford Gifford, probably saved the eye and preserved vision through the superotemporal quadrant of the cornea. It is also noteworthy that in the 2 cases in which grafts were not done what little healing did take place occurred through superficial vascularization in a conjunctival leash that invaded the cornea. These 2 cases indicate that a graft should be made in cases of lye burns in which recovery may occur without a graft, since the eyes with transplanted grafts heal more promptly and with increased vision, less corneal opacity and fewer sequelae.

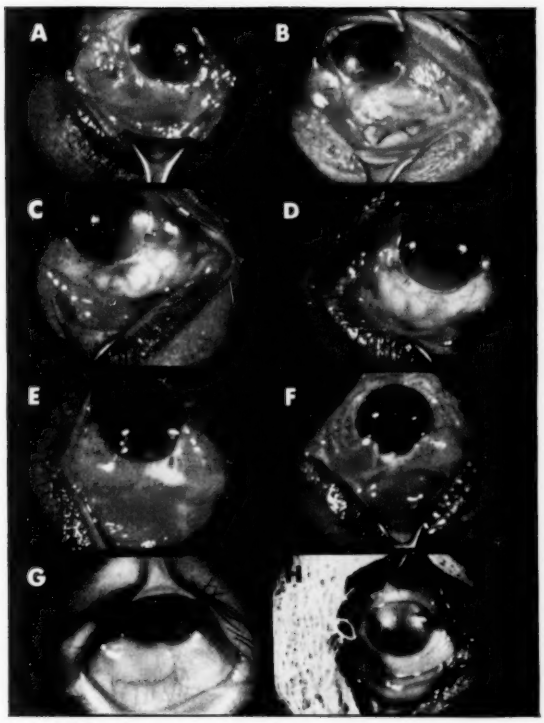
TECHNIC OF GRAFTING

The necrotic conjunctiva and episcleral tissue were excised, and the sclera was scarified with a scalpel. With a large lip clamp and a cataract knife, ample buccal mucous membrane was then taken. The buccal wound was not sutured. The graft, trimmed of submucous tissue, was applied directly to the denuded area and the margin of the graft sutured by interrupted no. 0025 Deknatel black silk to the intact healthy conjunctiva and the episclera (figure, *B*). When the entire bulbar conjunctiva was burned two horseshoe-shaped circumcorneal grafts were used. Sterile petrolatum was applied in a thin film over the graft. Gauze fluffs were applied under slight pressure and secured with adhesive tape to immobilize, but not to impair, the circulation. Over this dressing was placed a binocular bandage. Secretions were cleansed daily, and an ointment containing 1 per cent atropine was applied. The binocular bandage was removed on the fifth day, and sutures were removed on the seventh day.

REPORT OF CASES

Case 1.—H. Y., a Negro aged 39, was admitted on April 28, 1943, several hours after he had been greeted by his "girl friend" with a bucketful of lye thrown into the face and eyes. Vision in the right eye was reduced to counting fingers at 2 feet (60 cm.). There was pronounced necrosis of the palpebral and bulbar conjunctivas, with the typical "porcelain" appearance of the inferior half of the bulb, the process extending into the fornix. The lower three fourths of the cornea was dirty gray. Vision in the left eye was reduced to light perception. The entire bulbar conjunctiva was necrosed and dead white, with complete obliteration of blood vessels. The cornea was opaque gray. On April 28, ten hours after the accident, a graft of buccal mucous membrane was made in both eyes. The graft took promptly in the right eye, and on the fifth postoperative day the corneal opacity of that eye appeared to be receding. At the end of two weeks the cornea was clear except for a small, punctate-staining infiltrate at the inferior part of the limbus. The graft of the left eye sloughed completely. There was necrosis of the entire palpebral and bulbar conjunctiva, the cornea and the sclera. This eye, in spite of heroic medical and surgical measures, went on to complete necrosis, with perforation and sloughing of the entire cornea on May 13. The left eye was eviscerated, and the left bulb healed, with adhesions between the scleral stump and the lids, a palpebral fissure slit of 2 cm. remaining. Vision in the right eye returned to 4/10 after three and a half months. The entire cornea was clear except for a hazy rim of 3 mm. at the limbus from 5:30 to 9:30 o'clock (figure, *A*).

Case 2.—E. T., a Negro aged 20, was admitted on May 12, 1943, one hour after he had sustained burns of the eyes and face with lye as a result of a social accident. Vision in each eye was reduced to counting fingers at 2 feet (60 cm.). There were extensive second degree burns of the eyelids and face. The palpebral and bulbar conjunctiva of each eye showed advanced necrosis. The nasal and inferior quadrants of the bulb of the right eye had a porcelain white appearance, with obliteration of the blood vessels, and the nasal and nasal inferior portions of the right cornea were opaque gray as a result of direct burn (figure, *C*). The nasal, temporal and inferior quadrants of the left bulb were porcelain white, and the inferior three fourths of the left cornea was also opaque gray (figure, *D*). Buccal mucous membrane grafts were performed bilaterally nine hours after the injury. On the sixth postoperative day the corneas were clear except for a narrow rim inferiorly. The grafts took, and vision in each eye



A (case 1), buccal mucous membrane graft of the right eye three weeks after operation; *B*, buccal mucous membrane graft in place with interrupted black silk; *C* (case 2), lye burn in the right eye one hour after social accident; *D* (case 2), lye burn of the left eye one hour after the accident; *E* (case 2), buccal mucous membrane graft of the right eye three weeks after accident; *F* (case 2), buccal mucous membrane graft of the left eye three weeks after accident; *G* (case 5), buccal mucous membrane graft of the right eye two weeks after operation, and *H* (case 8), lye burn of the right eye six hours after the accident.

returned to 5/10 after three weeks (figure, *E* and *F*). Selective Service then claimed the patient.

CASE 3.—H. G., a Negro boy aged 7 years, was admitted on May 6, 1943 with an extensive gasoline burn of the left eye and the left side of the face. Vision in the left eye was reduced to perception of hand movements at 2 feet. Vision in the right eye was 20/20. There was a horseshoe-shaped ring of "porcelain" necrosis around the left limbus superiorly. The entire cornea stained deeply with fluorescein. On May 11 a horseshoe-shaped buccal mucous membrane graft was made. On the twelfth postoperative day there was slight clearing of the cornea at the periphery. One month after grafting, vision in the left eye had improved to 1/10. The superior half of the cornea was obscured by a gray opacity, and the lower half of the cornea showed multiple radial striate opacities. The graft was elevated and pink and measured 22 by 9 mm. in its greatest diameter.

CASE 4.—Data on this case were furnished by Dr. Sanford Gifford and Dr. C. E. Jaeckle. L., a white man, was admitted on June 7, 1943, to Passavant Memorial Hospital in the service of Dr. Sanford Gifford for a mucous membrane graft. The patient had suffered a sulfuric acid burn of each eye as the result of a battery explosion one week previously. Vision in the left eye was reduced to 8/40 and that in the right eye to 8/10. The lower half of the left cornea was cloudy, and there was a necrotic, white, avascular area below the limbus. On the fifth day after a buccal mucous membrane graft to the left eye, it was noted that the cornea appeared to be clearing. Two months after the injury the left cornea was entirely clear, and vision in the left eye had returned to 20/25 — 1.

CASE 5.—L. Z., a white boy aged 9 years, was admitted on Aug. 27, 1942, three days after a playmate threw lime into his right eye. Vision in the right eye was reduced to light perception and projection alone, and that in the left eye, to 1/10. The right cornea was milky gray and stained with fluorescein, and the bulbar conjunctiva from 5 to 8 o'clock was necrotic and porcelain white. The left cornea was clearing with the limbal circulation intact. A buccal mucous membrane graft was made in the right eye on August 29. On the fifth day after operation it was noted that the right cornea was less hazy, with the graft well vascularized. Six weeks after the injury the corneas were clear, and vision was 8/10 in the right eye and 10/10 + 3 in the left eye. The result in the right eye two weeks after operation is shown in *G* of the figure.

CASE 6.—J. F., a Negro man aged 46, was admitted on Feb. 19, 1943, two hours after his wife threw boiling water and lye into his eyes. Vision was reduced to 3/10 in the right eye and to 1/10 in the left eye. The lower third of the cornea of each eye was gray and opaque, and the perilimbal circulation was intact. On February 24, a rim measuring 2 mm., which appeared chemotic and pale, was noted below the limbus of the left eye from 5 to 8 o'clock. The viability of the circulation was equivocal; the question of a mucous membrane graft was debated, and the operation was not done. The cornea of the right eye responded slowly, but the process in the cornea of the left eye ran a stormy course. On March 3 the lower two thirds of the left cornea showed a deep opacity, and on March 9 a large corneal ulcer, 3.5 by 1.5 mm., developed 3 mm. inside the limbus, at 6:30 o'clock. This lesion healed with a sector-shaped, deep opacification of the cornea involving all layers of the stroma, with folds in Descemet's membrane. On June 1 corrected vision was 5/10 + 1 in the right eye and 4/10 in the left eye. The right eye healed, with a corneal opacity, measuring 3 mm., at 7 o'clock and a nebula which encroached on the center of the cornea.

The left eye finally healed, with a sector-shaped, deep corneal opacity involving the entire inferonasal quadrant of the cornea. A leash of conjunctival vessels at 6 o'clock invaded the cornea for 5 mm., through a superficial conjunctival shelf.

CASE 7.—O. Z., a Negro aged 41, was referred by the surgical service on May 8, 1943, one day after the left eye, the left side of the chest and the left arm were burned with hot lye thrown by his wife. Vision in the left eye was reduced to perception of hand movements at 2 feet. There was a collar of paleness inferiorly at the limbus, with intact perilimbal vessels; and the left cornea was milky and stained diffusely with fluorescein. Since the perilimbal circulation was intact, a mucous membrane graft was not made. The cornea began to clear peripherally, but infected infiltrates appeared at the edge of the central opacity, and a delimiting keratotomy was performed in the superotemporal quadrant on May 17. With therapy consisting of atropine, mercury bichloride and heat, the corneal lesion responded slowly. On June 12, vision in the left eye was only 1/10 with correction, and a diffuse, opalescent gray opacity involved the entire cornea except for a clear quadrant superotemporally, where a hard, gray, linear scar, measuring 4 mm., separated opaque from clear cornea. At 6:30 o'clock, and converging to a point 2 mm. inside the limbus, a wedge of superficial blood vessels invaded the cornea.

CASE 8.—W. W., a Negro aged 22, was referred by the surgical service on June 21, 1943, for severe burns of the face, eyes and mouth with lye. The patient's wife, six hours previously, had thrown a bucketful of hot lye into his face while he was asleep. The left eye prior to grafting is shown in *H* of the figure. Owing to the extensive burns of the mouth, it was impossible to obtain buccal mucous membrane for grafting. Prepuce grafts to each eye were made six hours after the injury. The left eye, which suffered total destruction of the perilimbal circulation, went on to complete necrosis and corneal perforation. The graft of the right eye took. However, there developed a virulent infection of the face and the right eye with Bacillus pyocyaneus, and the graft sloughed in part.

COMMENT

Prompt grafting with mucous membrane should be performed in cases of burns of the eye of the following types: (1) direct burn of the cornea with local or complete destruction of the perilimbal circulation; (2) direct burn of the cornea with a perilimbal circulation, the intactness of which is open to question, and (3) burn of the bulb with local destruction of the perilimbal circulation, unaccompanied by corneal involvement.

Early grafting stimulates vascularization, which provides nourishment to the cornea and is responsible for metabolic restoration in the cornea, with early return of functional power. Timely

grafting also prevents formation of scar tissue with symblepharon. If grafting is done too late or is omitted, local anoxemia leads to infiltration of the cornea and leukoma, with sequelae. Necrosis of tissue may give rise to tragic consequences. The observation of a porcelain white sclera offers a grave prognosis unless prompt grafting is performed. Even so, as occurred in 2 cases, the necrosis may be so extensive that grafting is of no avail, and the eye goes on to corneal perforation. Except when the entire perilimbal circulation is totally and irreversibly destroyed, early grafting not only shortens the course of the healing process but offers a better prognosis.

CONCLUSIONS

The results, based on 5 cases in which grafts of buccal mucous membrane were made and on 3 cases in which such grafting was not done, indicate the urgency of early grafting in treatment of ocular burns.

Every conjunctival or corneoconjunctival burn of real gravity should have a graft of buccal mucous membrane as soon as possible after the injury. The criterion for determination of the necessity and urgency of a graft is the destruction of the vascular supply to the cornea.

Buccal mucous membrane is the graft of choice in the treatment of severe ocular burns. Preserved heterogenous conjunctiva and cadaver conjunctiva offer future possibilities but require further clinical trial.

Immediate transplantation of buccal mucous membrane should be done in cases of burns of the eye due to war gases, which cause vascular interference in the perilimbal circulation. The lessons learned in civilian life with buccal mucous membrane grafts may prove of value in prevention of the delayed effects of war gases on the cornea.

The late Dr. Sanford R. Gifford, attending ophthalmologist at Cook County Hospital, made the original suggestion for this work and offered valuable criticism. Dr. Isadore Siegel translated the long German monograph by Thies.

111 Virginia Avenue.

TREATMENT OF METASTATIC MENINGOCOCCIC ENDOPHTHALMITIS

REPORT OF A CASE

ARLINGTON C. KRAUSE, M.D., AND WILLIAM ROSENBERG, M.D.
CHICAGO

Ocular complications of cerebrospinal meningitis are less frequent than they were in years past. Prompt use of meningococcic antitoxin and extensive use of sulfonamide compounds have been responsible for the great reduction in the number of complications. The gravest, most important ocular complication is metastatic endophthalmitis, terminating usually in the formation of an abscess in the vitreous, with subsequent organization, in the formation of a cyclitic membrane and frequently in complete retinal detachment. Endophthalmitis occurs in about 5 per cent of all the cases in epidemics. Lazar [1] cited 30 instances in 575 cases, or 5.2 per cent. Bilateral involvement was present in 9 cases in his series. Lewis [2] reported an incidence of 5.7 per cent in 350 cases, with involvement of both eyes in 4 instances.

Epidemic meningitis is primarily an infection of the blood stream, with localization in the meninges and other organs occurring secondarily. Dunphy [3] stated: "In almost all cases the ocular inflammation begins during the stage of bacteremia or shortly after the meningeal symptoms have made themselves evident. Occasional late involvement of the eyeball occurs, and this is probably due to septic emboli." According to Lewis the earliest occurrence of endophthalmitis was on the second day of the disease and the latest on the twelfth. The average occurrence was on the fifth day. Lazar, in 4 case reports, indicated that involvement of the vitreous was present in all patients on their admission to the hospital. In the first case it was noted twenty-four hours after the onset of the illness and in the second three days afterward; in the third and fourth cases the children were admitted primarily because of the ocular infection. The diagnosis of epidemic meningitis in the latter case was made only after examination of the blood and the spinal fluid. Meningococci have been cultured from within the eyeball. Pathologic changes in eyes enucleated for meningococcic endophthalmitis include no changes that are pathognomonic and differ in no way from those resulting from endophthalmitis from other sources. Inflammation of the anterior portion of the uvea and the choroid, retinitis, usually with detachment, and abscess of the vitreous with organization are common in almost all cases.

The visual end results of endophthalmitis usually are disastrous. However, Lazar reported 2 cases and Lewis [4] 1 in which complete recovery occurred, with return of normal vision. In Lazar's first case, beginning bilateral endophthalmitis was diagnosed one day after the onset of illness. The anterior chambers and the vitreous were hazy, but apparently no frank abscess or membrane formation was noted. Treatment consisted of intravenous administration of meningococcus antitoxin, and clearing of the eyes was noted in twenty-four hours, with complete recovery in two days. Similarly in the second case bilateral endophthalmitis was diagnosed when the patient was admitted to the hospital three days after the onset. Treatment was instituted immediately with intravenously administered meningococcus antitoxin. In addition, intramuscular injections of milk and subconjunctival injections of mercuric oxycyanide were given. One eye recovered with normal vision, and the other underwent phthisis bulbi.

Lewis reported 1 instance of recovery. In his patient the vitreous was filled with grayish opacities, the iris was not involved and there was no ciliary injection. The opacities absorbed rapidly in one week and completetly disappeared. Lewis stated that this condition probably should not be classed as endophthalmitis since apparently only the vitreous was involved. Antimeningococcus serum was given intravenously and intraspinally the day the symptoms first appeared. The injection of antimeningococcus serum or meningococcus antitoxin intraocularly and sub-

From the Division of Ophthalmology, Department of Surgery, the University of Chicago.

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conjunctivally has been tried by several observers, with apparently no definite results.

The more modern treatment of meningococcic meningitis is with antitoxin given intravenously and sulfonamide compounds administered enterally and parenterally.

REPORT OF A CASE

Miss J. W., aged 18 years, was admitted to Municipal Contagious Disease Hospital on July 11, 1943. At that time she was suffering from meningitis, which had a history of four days' duration. Her temperature was 101.8 F. taken rectally, her pulse rate, 120 and her respiratory rate 26. She was irrational. Petechiae were numerous over the body and especially on the extremities. Both eyes were severely injected, and the corneas appeared cloudy. It was difficult to determine whether vision was present. The ocular condition was diagnosed endophthalmitis.

The white cell count of the blood was 59,800, and the red cell count was 3,400,000. No growth was obtained from a blood culture. A lumbar puncture was not made.

Treatment consisted of intravenous administration of 40 cc. of antimeningococcus serum in 1,000 cc. of 5 per cent dextrose in saline solution within a few hours of the patient's admission to the hospital. This was followed eight hours later by 5 Gm. of sodium sulfadiazine given intravenously. Beginning on July 12, at 2:00 a. m. two more doses of 2 Gm. of sodium sulfadiazine were given intravenously four hours apart. After that 1 Gm. of sulfadiazine was given orally every four hours until July 23. On July 14 the blood level of sulfadiazine was 19.3 mg. per hundred cubic centimeters.

During the first few days the eyes were irrigated with boric acid solution every hour except at night. On July 14 ophthalmic ointment was applied to the eyes. At that time the patient's condition was poor; her temperature was 103 F., her pulse rate 120 and her respiratory rate 24. On this same day the patient complained a great deal of painful joints, which had been tender at the time of admission. She also stated that she was unable to see. On July 15 a solution of 20 per cent mild protein silver was used for eye drops. Two drops of 1 per cent solution of homatropine hydrobromide was used for each eye. On July 16 10 grains of acetylsalicylic acid was given on two occasions. On July 17 a 1 per cent solution of atropine sulfate was instilled in each eye, and this treatment was used periodically during the remainder of the patient's illness. Fifteen grains of sodium salicylate with 5 grains of sodium bicarbonate was given twice daily from July 22 until July 26. Hot compresses were repeatedly applied to the eyes.

By July 25 the patient's temperature had returned to normal, and her general condition was much improved. Her eyes were also less injected than at the time of admission to the hospital, but it seemed doubtful if vision existed.

The patient was released from Municipal Contagious Disease Hospital on July 30, 1943. On that date the temperature was normal, as it had been for five days previously. Although the eyes appeared to be improved, it was difficult to determine whether or not there was perception of light. There was nothing significant in regard to any of the reports of urinalyses and a final examination of the urine on July 30 revealed normal conditions except for the presence of some albumin.

On July 30, 1943 she was admitted to the medical isolation service of Dr. C. Phillip Miller at the Albert Merritt Billings Hospital, the University of Chicago. Treatment was begun with 3 Gm. of sulfadiazine, followed by 1 Gm. every four hours. Examination of the eyes at that time revealed vision in each eye reduced to perception of hand movements at about 3 feet (90 cm.), and also intense bilateral photophobia, severe mixed bulbar injection and extreme ciliary tenderness. The corneas and the anterior chambers were extremely hazy and the pupils were irregularly dilated and adherent to the lenses. A yellow dense exudate was seen behind each lens, so that details of the fundi could not be seen. Markings on the irises were obliterated, and dull red reflexes were obtained from the pupillary spaces on transillumination. The diagnosis was metastatic meningococcic endophthalmitis of the right and the left eye. Instillation of 1 per cent solution of atropine sulfate three times a day in each eye and typhoid fever therapy were begun. Although no blood cultures were obtained at Municipal Contagious Disease Hospital, on July 3, 1943 the patient's blood serum agglutinated type I meningococci in titers of 1:32. There was no agglutination with type II or type II *a*. It was presumed that the infection must have been with type I.

After consultation with Dr. Miller it was decided to treat the patient with penicillin administered by iontophoresis. The penicillin was produced by E. R. Squibbs & Sons (25,000 Florey units per ampule) and was supplied [5] by the Committee on Medical Research of the Office of Scientific Research and Development by arrangement with Dr. Chester S. Keefer. The solution was made up in 0.9 per cent solution of sodium chloride so as to contain 1,000 units per cubic centimeter.

Two drops of a solution of penicillin containing 1,000 Florey units per cubic centimeter was instilled into the conjunctival sacs. The lids were closed and negative electrodes placed over the closed lids with a positive electrode placed over the neck. Two milliamperes of current was allowed to pass for five minutes in each eye. This procedure was repeated on July 31 and Aug. 1. From August 2 to August 24 a current of 3 to 4 milliamperes was allowed to pass for ten minutes in each eye. She received typhoid fever therapy on July 31 and on August 14; the reaction was a moderate fever. In addition, the sulfadiazine therapy was continued until Aug. 4.

Examination of her eyes was extremely difficult, because of excessive photophobia and poor cooperation. The fundi were examined with the patient under vinethene anesthesia, on August 9, 16 and 26. On August 9 both eyes showed a severe anterior uveal reaction, with distinct conjunctival injection. The corneas were extremely hazy; the anterior chambers were cloudy; there were complete posterior synechia, as before, early seclusio and occlusio pupillae, pro-

5. The penicillin was provided by the Office of Scientific Research and Development from supplies assigned by the Committee on Medical Research for clinical investigations recommended by the Committee on Chemotherapeutic and Other Agents of the National Research Council.

nounced atrophy of the irises and proliferation of pigment. The right fundus showed a red, granular reflex uniformly. A small white cloud was present over the disk. The vitreous was extremely hazy, and no details could be seen. The left fundus showed a yellow reflex, with a questionable suggestion of a pink reflex below. On August 16 the media of the right eye showed some clearing, so that the retinal vessels could be recognized faintly. The left eye still showed no red reflex. The external reaction continued to be severe in both eyes. On August 26 there was no change in the left eye, but the right eye began to show evidences of clearing, externally and internally. The bulbar reaction was less intense, and the retina showed more clearly. A light white haze was seen on the disk between 3 and 6 o'clock and on the adjacent retina about ½ papilla diameter from the area. The macula was seen, but no foveolar reflex could be made out.

The patient was discharged from the hospital on September 2. She was seen in the outpatient ophthalmic clinic on September 18. Her mother stated that gradual improvement had occurred while she was at home, so that the patient's eyes had gradually become white and she had begun to open them spontaneously. Photophobia had almost completely disappeared; she walked around the house unaided and was able to perform simple tasks with ease, such as washing dishes. Vision in the right eye was 20/200 as determined by the Snellen chart, and she could distinguish hand movements with the left eye. It was believed that she could probably see more than the chart indicated.

On October 29 the patient was seen again in the outpatient clinic. The mother reported that she had improved remarkably well, so that she could carry on all of her normal activities. There was no voluntary or involuntary blepharospasm, the eyes remained completely white and there was no indication of any discomfort. Vision in the right eye was 20/50 + 2 as measured on the Snellen chart and 20/30 when tested with the number chart. She could only distinguish hand movements with the left eye. The right eye was white; there was no evidence of any active inflammation; the pupil was irregularly dilated and completely adherent to the lens. The cornea was clear. The tactile tension was normal. Slit lamp examination of the anterior segments was not possible because of poor cooperation, although examination with the ophthalmoscope revealed no gross lesions of the retina. The disk and the vessels appeared normal, and the macular area, although not well seen, also looked normal. There was a slight generalized haze of the vitreous, but no discrete opacities were seen. The left eye revealed a clear cornea and a large, irregularly dilated pupil, completely adherent to the lens. A dense white opaque mass occluded the pupillary space. The tactile tension was slightly less than that of the right eye.

COMMENT

The patient on discharge from the hospital after treatment for meningococcic infection had bilateral metastatic endophthalmitis, leading apparently to total blindness. She was again admitted to the hospital for treatment with penicillin. Penicillin was chosen because it is bactericidal and bacteriostatic and is superior to the sulfonamide drugs in that its activity is not inhibited by large numbers of bacteria or by the presence of pus or autolysates of tissue. Furthermore, treatment with sulfadiazine caused no evident improvement of the ocular disease. No report on the use of penicillin for clinical meningococcic infection was found in the literature. However, Hobby, Meyer and Chaffee [6] found that meningococci were susceptible in vitro to the bactericidal or bacteriostatic action of penicillin.

The choice method of administration of penicillin is intravenous or intramuscular injections for generalized infections and local applications for focal infections.[7] Frequent small doses are better than less frequent larger doses, since the tissues require a constant concentration. Treatment is continued until clinical signs have disappeared.[8]

Because of the small quantity of penicillin which was available and because of the localization of residual meningococcic infection a decision was made to try iontophoresis. Recently Boyd,[9] using sodium sulfathiazole, and von Sallmann,[10] using scopolamine hyrobromide and atropine sulfate, found that a decided increase in the concentration of the drugs in the cornea and the aqueous humor may be obtained by means of iontophoresis. Von Sallmann [11] later made similar observations after trying sulfadiazine and penicillin on experimental animals.

In those cases in the literature in which recoveries were reported ocular complications were present and diagnosed early, i. e. within the first five days, and treatment was begun immediately. Cure was attributed to the specific general and not local therapy. There were no instances of recovery after the pathologic process seemed to be well established. Indeed treatment was not directed specially to the ocular complications, but these responded to treatment of the

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10. von Sallmann, L.: Iontophoretic Introduction of Atropine and Scopolamine into the Rabbit Eye, Arch. Ophth. **29**:711 (May) 1943.

11. von Sallmann, L.: Penicillin and Sulfadiazine in the Treatment of Experimental Intraocular Infection with Pneumococcus, Arch. Ophth. **30**:426 (Oct.) 1943.

meningococcemia as a whole. The clinical application of iontophoresis of penicillin as a means of attaining a more rapid and concentrated penetration of the drug in the eye, as presented in this case, is evidently well worth a trial.

SUMMARY

The treatment of metastatic meningococcic endophthalmitis after recovery of the patient from meningitis has been considered hopeless. A patient with meningococcic endophthalmitis was treated with sulfadiazine given by mouth and penicillin applied by ocular iontophoresis. There is reason to believe that penicillin and sulfadiazine were the effective agents producing recovery from unilateral endophthalmitis in our patient. Recovery was remarkable in that the disease process was present without remission for twenty-four days before treatment was begun. There was no response to the usual therapeutic methods, and favorable prognosis appeared hopeless.

STRABISMUS IN ADULTS

ANALYSIS OF OPERATIVE RESULTS IN SIXTY-FIVE CASES

MAJOR IRVING I. SHURE
MEDICAL CORPS, ARMY OF THE UNITED STATES

The importance of proper treatment for strabismus has been emphasized by Peter,[1] Wilkinson [2] and Davis.[3] A review of the recent literature on strabismus shows that inadequate emphasis is placed on the necessity for proper treatment of adults so affected. The feeling of inferiority among these patients is inconceivable. One cannot appreciate the timidity, the lack of initiative and the complete introversion of these otherwise intelligent people unless one has had the opportunity of treating a large number. I report here an analysis of results in 65 cases of strabismus in adults, all of whom underwent operative procedures and received orthoptic treatment. The results obtained in this series, from the standpoint both of the cosmetic effect and of functional recovery, convince me that too many ophthalmologists have developed an attitude of defeatism toward the treatment of this condition. Much can be accomplished for patients with this disorder, and it is hoped that this analysis will lead others to pursue a more aggressive course of treatment.

A proper diagnosis of the type of strabismus present is equally as important in adults as in children. Before treatment is instituted in any case, the following procedures should be carried out: (1) a complete history, including date of onset and the presence of a familial tendency, should be obtained; (2) refraction should be done; (3) the amount of deviation should be estimated; (4) the near point of convergence should be determined; (5) the degree of fusion, if any, should be measured, and (6) the presence or absence of anomalous retinal correspondence should be noted. This routine was followed in the series of cases studied.

An accurate history is essential, and its importance has been stressed by Lordan [4] and Guibor.[5] The age at onset and the presence or absence of a hereditary tendency are of the utmost importance in arriving at a proper diagnosis. The appearance of a strabismus soon after birth, or at 6 months of age, indicates a nonaccommodative type. Visual acuity is usually 20/20 in both eyes. Complete absence of the fusion faculty was demonstrated in patients with strabismus by Peter [6] and Worth.[7] Burri [8] did not admit the presence of a specific fusion center but expressed the belief that fusion is an act of learning, comparable to that of walking or talking. I am in agreement with Peter and Worth with respect to the presence of a fusion center and am convinced of its importance in the causation of strabismus. The absence of a fusion faculty predicates a poor prognosis. But if fusion is present in any degree, it can be developed in persons well past middle adult life (Peter [6]).

Refraction should be done under full cycloplegia. All patients in this series were examined under cycloplegia induced with 1 per cent homatropine hydrobromide, several drops being administered every ten minutes for six doses. The degree of strabismus was noted and recorded before and during the cycloplegia. Postcycloplegic examination was performed ten days later. Fundoscopic examination revealed the presence of any ocular disease.

The degree of deviation was estimated by two methods. When practical, the screen test was utilized, and the movements of both eyes were neutralized by means of prisms. A state of suppression, amblyopia or poor fixation is common in adult patients with strabismus. Davis and Sheppard [9] have shown that the screen and prism test is useless with such patients and that one

1. Peter, L. C.: Technic of Orthoptic Training in Squint, Arch. Ophth. **14**:975-984 (Dec.) 1935.
2. Wilkinson, O.: Early Treatment of Squint, South. M. J. **34**:844-846 (Aug.) 1941.
3. Davis, W. T.: Ocular Motility Then and Now, South. M. J. **36**:228-234 (March) 1943.
4. Lordan, J. P.: Orthoptic and Surgical Management of Strabismus, Arch. Ophth. **12**:843-849 (Dec.) 1934.
5. Guibor, G. P.: Early Diagnosis and Non-Surgical Treatment of Strabismus, Am. J. Dis. Child. **52**:907-915 (Oct.) 1936.
6. Peter, L. C.: Extra-Ocular Muscles, ed. 3, Philadelphia, Lea & Febiger, 1941, pp. 216-218.
7. Worth, C.: Squint, ed. 5, Philadelphia, P. Blakiston's Son & Co., 1921, p. 78.
8. Burri, C.: Process of Learning Simultaneous Binocular Vision, Arch. Ophth. **28**:235-244 (Aug.) 1942.
9. Davis, W. T., and Sheppard, E.: Interpretation and Evaluation of Tests for Strabismus, South. M. J. **34**:559-565 (June) 1941.

must resort to the perimeter. This instrument was used in every case in this series, usually in addition to other means of strabismometry. The strabismus was measured for 20 feet (6 meters) and for 13 inches (33 cm.).

The importance of a proper evaluation of the existing status of the near point for convergence in patients with strabismus was stressed by Jameson.[10] He maintained that the condition of the

Fig. 1.—*A*, divergent strabismus in the right eye, with a deviation of 35 degrees. Visual acuity was 20/20 in both eyes. *B*, result obtained by recession and advancement of the internal rectus muscle and recession of the external rectus muscle. Fusion grade 3 was obtained.

musculature, or, rather, its innervational status, can be determined by this procedure. White[11] placed great importance on determination of the deviation for 20 feet and for 13 inches. If the case is one of convergent strabismus and the deviation is greater for distance than near vision, the condition is recorded as divergence insufficiency. If the deviation is greater for near vision, convergence excess is present. In the case of divergent strabismus, if the deviation is greater for distant than for near vision, divergence excess is present, and if the deviation is greater for near vision, convergence insufficiency exists. Wilkinson[12] divided convergent strabismus into the static and the dynamic type. Static convergence is equal to the squint. The patient fixes an object with his straight eye. The object is gradually brought toward the eye, and the movements of the deviating eye are noted. The point at which the deviating eye corrects itself is the near point. If, when the object is brought closer to the eye, the convergence is increased, an excessive dynamic convergence, or convergence excess, is indicated. If the eyes tend to correct themselves without further convergence, a divergence insufficiency exists. A study of the results obtained in the present series of cases by these methods shows that the two procedures arrive at the same conclusion in somewhat different ways. The type and extent of the operative procedure to be employed can be determined by the results obtained from these tests.

The ultimate aim in any method of treatment for strabismus is ability to obtain and maintain stereopsis. Feldman and Taylor[13] noted three grades of binocular vision: (1) binocular macular perception, (2) superimposition of images and (3) stereopsis. Burian[14] defined binocular macular perception as the visual state in which both retinas are stimulated simultaneously at points having the same innate, or common, visual direction. The two images falling on the two foveas are then converted into one image; thus, only one object is seen. This superimposition, or the combining of two images into one, is known as fusion, or the second stage. Fusion precludes the presence of a properly functioning fusion faculty. Determination of the presence and degree of fusion is most important in all

Fig. 2.—Alternating strabismus, with a deviation of 43 degrees. Visual acuity was 20/20 in both eyes. *B*, result of resection and advancement of the internal rectus muscle and recession of the external rectus muscle of both eyes. Fusion grade 3 was obtained.

cases of strabismus, both from a prognostic and from a therapeutic standpoint. A stereoscope of

10. Jameson, P. C.: Entity of Muscle Recession, Arch. Ophth. **21**:362-370 (Feb.) 1939.

11. White, J. W.: The Importance of a Complete Diagnosis to a Successful Treatment of Strabismus, Surg. Gynec. & Obst. **74**:565-566 (Feb.) 1942.

12. Wilkinson, O.: Strabismus: Its Etiology and Treatment, ed. 1, St. Louis, C. V. Mosby Company, 1927, pp. 135-136.

13. Feldman, J. B., and Taylor, A. F.: Obstacles to Squint Training—Amblyopia, Arch. Ophth. **27**:851-868 (May) 1942.

14. Burian, H. M.: Fusional Movements in Permanent Strabismus, Arch. Ophth. **26**:626-652 (Oct.) 1941.

standard make, with use of the cards devised by Wells, affords an ideal and inexpensive instrument for the measurement of fusion. This instrument was used in all cases reported in this series.

The presence or absence of anomalous retinal correspondence was determined in each case by means of the after-image test, as advocated by Bielschowsky.[15]

Fig. 3.—*A*, convergent concomitant strabismus in the left eye, with a deviation of 18 degrees. Visual acuity was 20/20 in the right eye and 20/100 in the left eye. *B*, result obtained by resection of the external rectus muscle and recession of the internal rectus muscle. No fusion or improvement of vision resulted.

ANALYSIS OF CASES

A total of 65 operative cases is reported in this study. All the patients were men, in good health. A review of the observations reveals the following interesting facts:

Age.—The youngest patient operated on was 18 years of age, and the oldest, 39. The average age for the entire series was 27 years.

Degree of Deviation.—As stated in the first part of this paper, I found the perimeter an excellent instrument for measuring the amount of deviation. In the 51 cases of convergent strabismus the lowest amount of deviation was 8 degrees, and the highest, 45 degrees. In 2 cases between 5 and 10 degrees, in 12 cases between 1 and 20 degrees, in 23 cases between 20 and 30 degrees and in 4 cases over 40 degrees of deviation was present. Analysis of the cases of divergent strabismus showed deviations of between 20 and 30 degrees in 6 cases and of between 30 and 40 degrees in 6 cases. The left eye was the squinting eye in 25 of the cases of convergent strabismus, and the right eye was the offender in the remaining 26 cases. Analysis of the cases of divergent strabismus showed that the squint was in the left eye in 5 cases and in the right eye in 7 cases.

Refractive Error.—In practically all cases in this series refraction had been carried out at various times, and refractive errors had been prescribed for prior to examination at the station hospital. In all patients, however recent the last refraction, examination was again made under full cycloplegia. Of the 65 cases studied, no refractive error was present in 31. Interestingly, into the latter group fell a number of cases with deviations of 40 degrees or over. No definite relation could be shown between the degree of deviation and the refractive error, an observation corroborating that of Dunnington and Wheeler.[16] In 2 cases there were refractive errors of over 3 D. In 4 cases of convergent strabismus there was minimal myopia. In 5 cases of convergent strabismus the squint was of the accommodative type. In the last-mentioned cases the wearing of the corrective lenses reduced the amount of deviation from 4 to 20 degrees.

Amblyopia and Visual Acuity.—Amblyopia was taken to mean the missing of the first three lines on a standard Snellen chart or vision below 20/50. Cases were eliminated in which the fundus was pathologic. There were 12 cases of amblyopia, in 7 of which refractive errors above 2.5 D. were present. In the remaining 3 cases no refractive error was noted. This

Fig. 4.—Convergent concomitant strabismus, with deviation of 28 degrees in the right eye. Visual acuity was 20/40 in the right eye and 20/20 in the left eye. *B*, result of resection and advancement of the external rectus muscle and recession of the internal rectus muscle. Fusion grade 2 was obtained.

15. Bielschowsky, A.: Lecture on Motor Anomalies: IV. Etiology of Strabismus, Am. J. Ophth. **21**:329-342 (Dec.) 1938.

16. Dunnington, J. H., and Wheeler, M. C.: Operative Results in Two Hundred and Eleven Cases of Convergent Strabismus, Arch. Ophth. **28**:1-11 (July) 1942.

indicates a definite relation between refractive error and amblyopia, probably through suppression of the squinting eye.

Type of Operation.—The type of operation to be performed in each case was determined only after a review of the clinical data. If hyperirritability of the internal rectus muscle was present, i. e., convergence excess, a maximum recession was performed on that muscle. If, on the other hand, divergence insufficiency prevailed, the maximum operative effort was expended on a resection and advancement procedure of the external rectus muscle. More satisfactory results were obtained in cases in which a recession of one muscle was combined with a resection and advancement of the opposing muscle. I am in complete agreement with Jameson,[10] who stated that the internal rectus muscle is much more sensitive to recession than the external rectus muscle and that measured recessions are quite satisfactory. After a thorough trial, it was found that for each millimeter of recession or resection of the internal rectus muscle, approximately 3 degrees of deviation will be corrected. Likewise, each millimeter of operative effort on the external rectus muscle will yield about half this correction, or 1.5 degrees. If these figures are used as a guide in calculating the approximate procedure on each muscle prior to operation, a most satisfactory cosmetic result will obtain in the majority of cases. This statement is made with full realization of the individual variations and anatomic differences that an operator invariably encounters.

TECHNIC

The method of tendon recession described by Jameson[10] and Wiener and Alvis,[17] with slight modification, was employed. A vertical incision is made over the insertion of the tendon of the muscle to be operated on. Good exposure is obtained by blunt dissection for a distance approximately equivalent to the diameter of the cornea. The capsule of Tenon is grasped below the tendon's lower border and carefully snipped, and the point of a tendon hook is introduced beneath the muscle. Care is taken to include all the muscle fibers on the hook. The point is then allowed to show through the opposite border by rotation of the hook toward the operator, and Tenon's capsule is again snipped. A second tendon hook is placed in the last opening made, and the fibers of the capsule of Tenon, the ligament of Lockwood and adjacent structures are all carefully dissected free. The bluish white sclera can be seen beneath. Two double-armed sutures of no. 0000 plain catgut, threaded in atraumatic needles, are used. The sutures are passed through the tendon from the scleral surface upward, one suture on each end, and placed as close to the scleral attachment as possible. The muscle is then severed from the sclera. The amount of recession having been previously computed, a point marking the new site of insertion on the sclera is made. The atraumatic needles are carefully teased through the superficial fibers of the sclera, on as broad a base as the area of original insertion. The sutures are then securely tied, and the conjunctiva is closed with an uninterrupted black silk suture. In the procedure of resection and advancement, the conjunctiva is opened and the muscle exposed in a manner similar to that for recession. A vertical incision is made over the tendon of the muscle involved, approximately 7 mm. back of the limbal margin, and the incision extended the length of the cornea. Tenon's capsule is carefully picked up at its lower border and snipped. The point of a tendon hook is introduced into the opening and carried beneath the muscle, care being taken to include all the muscle fibers. The hook is then brought against Tenon's capsule, which is snipped at its upper border, and a second tendon hook is placed into the latter opening. All fibers of Tenon's capsule are then carefully dissected free, and the entire muscle is exposed toward the equator of the globe by separating the two hooks. Two double-armed, atraumatic sutures of plain no. 0000 catgut are used. The amount of muscle to be resected or advanced having been carefully calculated by the micrometer, one of the double-armed sutures is inserted at the measured point into approximately the central portion of the muscle. The second needle of the same suture is then similarly placed near its lateral border. The latter suture is once more brought around the lateral margin of the muscle for additional fixation. The second suture needle is similarly brought through the opposite border of the muscle, as described. A firm grasp is taken on both sutures, which are held upward, and the muscle is severed. The excess muscle at the stump is cleanly removed down to the sclera. With firm fixation at the stump by means of forceps, the muscle is reinserted into the new point by carefully teasing the needle through the superficial layers of the sclera. Since the conjunctiva is firmly adherent at this point, it is well to see that clean dissection has been performed. Care must be taken to see that a firm bite is taken into the scleral fibers in this region. The muscle is carefully brought up to its new insertion and tied. A single running black silk suture is used to close the conjunctiva. Only the eye operated on is bandaged, the good eye being left open. The bandage is removed in twenty-four hours, and the usual postoperative chemosis is controlled with cold packs. The patient is placed under orthoptic training forty-eight hours after operation. Sessions begin with fifteen minute periods twice daily and are gradually increased to thirty minutes twice a day. The sutures are removed from the conjunctiva on the fifth day, and the patient is discharged to duty in ten days.

As previously stated, a surprisingly accurate estimate of the amount of muscle to be recessed, resected or advanced can be made in a large number of cases. The amount was based on the principle of a 3 degree correction for each millimeter of recession of the internal rectus muscle and a 1.5 degree correction for each millimeter of recession of the external rectus muscle. An ordinary micrometer was used at the operating table to determine the amount of the operative procedure. It will be found that observation of the general rule just stated will result in approximate parallelism of the eyes in a great majority of cases, especially if

17. Wiener, M., and Alvis, B.: Surgery of the Eye, ed. 1, Philadelphia, W. B. Saunders Company, 1940, pp. 355-356.

careful study has been given to the status of the muscle primarily involved.

Elimination of the Prince or Noyes muscle forceps has been achieved merely by substitution of sutures to fix the muscle. Use of the latter allows a clearer field, with better exposure of the operative site. Crushing of the muscle is also avoided.

The statement of Jameson [10] that "it is better to leave a slight convergence than to run the risk of an overcorrection," has not received confirmation from the results in this series. I have consistently found that patients with parallelism on leaving the table will show residual convergence when examined several months later. If, on the other hand, about 5 degrees of divergence is present at the time the operation is finished and the patient is placed under supervised orthopic training within forty-eight hours, not only excellent cosmetic results but various degrees of fusion will be obtained in a great number of cases. It is granted that the results obtained in the treatment of strabismus are extremely variable. In the majority of cases, however, results are satisfactory if fundamental principles are adhered to, evaluation of muscle power is properly performed and good surgical technic is employed.

The older method of bandaging both eyes for four to five days after an operation on the ocular muscles has been found necessary. It is now known that the eyes assume not only the position of rest beneath bandages, i. e., up and out, but that considerable movement takes place. Only the eye operated on was bandaged after each operation, and the bandage was removed in twenty-four hours. If care is taken at the time of operation to see that a good bite is taken into the scleral fibers, slipping of the suture need not be feared.

RESULTS

Two methods of analysis lie open in any review of strabismus treated by operation. Many surgeons are content to achieve a satisfactory cosmetic result. The value of such an achievement to the patient's psychologic makeup is unquestioned. Only the surgeon who has seen children, and even adults, made introverts by pronounced strabismus can appreciate the joy which the patient will express over a result that he often questions. The second objective, and certainly the one all surgeons treating abnormalities of the ocular muscles should strive for, is that of fusion and stereopsis. In each case in this series the patient's ability to attain fusion was evaluated prior to operation. In cases of amblyopia of course this possibility becomes remote. A good cosmetic result must, of necessity, suffice in a case of the latter defect.

An attempt was made, through careful individualization, to ascertain the type of operation most suited in the particular case. The 65 cases were broken down into four groups on the basis of the operative procedure. In 55 cases a recession combined with a resection and advancement of the opposing muscle was done. A shortening operation of one muscle only was performed in 2 cases while a recession of one muscle and a resection of the opposing muscle, with reinsertion at its original site, were performed in 2 cases. In the remaining 6 cases resection and advancement were performed on one muscle, with no operation on its antagonist.

In 14 of the 55 cases in which recession was combined with resection and advancement, undercorrection resulted. In 7 of these cases a second operation was required, and in 1 case four operative procedures were necessary. In the remaining 6 cases there was no further operative intervention, for one reason or another. Satisfactory parallelism of the eyes was obtained in all 7 cases in which the second operation was performed. It is significant that, though the methods of measurement of the squint and the operative figures remained the same, the cases in which there was a small primary deviation were the cases in which an undercorrection resulted.

Analysis of Results of Operative Treatment in Fifty-One Cases of Strabismus in Adults

Type of Strabismus	Number of Cases	Over-correction	Under-correction	Second Operative Procedure	Fusion: Final Result			
					None	Grade I	Grade II	Grade III
Convergent	51	0	12	5	25	5	7	14
Divergent	12	0	2	2	3	1	3	5
Alternating	2	0	0	0	0	1	0	1

An interesting fact revealed in this study concerns the behavior of divergent strabismus. In 12 cases of this type of squint recession combined with resection and advancement was employed. In no case did overcorrection result. The cosmetic results were excellent, although the deviations were by far the most extreme of those in the entire series. In the 2 cases in which recession of one muscle was combined with resection of the opposing muscle, deviations of 15 and 16 degrees were noted. Final results showed cure in both cases. Resection of one muscle resulted in production of a cure in the 2 cases in which this method was employed. The deviations were 8 and 15 degrees respectively. In all 6 cases in which resection and advancement of one muscle only were done deviations ranged from 8 to 18 degrees. A good cosmetic effect was obtained in all cases.

No patient in the entire series was considered cured until a thorough trial was made to develop fusion. An ordinary stereoscope was used, consisting of two lenses of + 5.25 D. each, separated by a distance of 8.5 cm. The method of the preliminary test advocated by Linksz [18] was used. It was found that the charts devised by Dr. Wells gave the best results in the development of fusion. Each patient was given orthoptic training on removal of the bandage, usually about forty-eight hours after the operation. Treatments consisted of two sessions of fifteen minutes each day for one week, after which the treatment periods were lengthened to thirty minutes. All exercises were supervised, and a daily record of the patient's progress was carefully noted.

The basis for the orthoptic procedure lies in the brain's abhorrence of diplopia produced by the stereoscope. As the patient looks into the instrument, the images tend to move together, since this is the only method by which the diplopia can be overcome. Because both macules are stimulated simultaneously, suppression of either image is virtually impossible. This presupposes, of course, the presence of binocular macular perception.

It has long been recognized that the presence of suppression, amblyopia, poor fixation or anomalous retinal correspondence constitutes obstacles in the development of fusion. An attempt was made in each case to break down the influence of these factors by means of flash illumination, mental reeducation and daily treatments with the stereoscope. Often, two or more of these conditions were present in 1 case. In some cases an obstacle of this kind proved refractory to all orthoptic training, and my associates and I were content to call a good cosmetic result a cure in several instances of this type.

A review of the results obtained with orthoptic training after operation was most gratifying. An average of twenty treatments was given each patient in this series. The smallest number of treatments given to one patient was twelve; the largest, thirty-two. These treatments were all given at daily intervals. The results were recorded as no fusion, fusion grade 1 (simultaneous binocular macular perception), fusion grade 2 (superimposition of images) and fusion grade 3 (stereopsis). In 28 cases no fusion developed after prolonged orthoptic training. Fusion grade 1 was obtained in 7 cases, fusion grade 2 in 19 cases and fusion grade 3 in 20 cases. When it is remembered that amblyopia was present at the start of the treatments in 12 cases, it can readily be seen that the results obtained were surprisingly good.

18. Linksz, A.: The Stereoscope as an Orthoptic Instrument, Arch. Ophth. **26**:389-407 (Sept.) 1941.

COMPLICATIONS

In no case in the entire series did postoperative infection result. The usual chemosis following operative trauma was easily controlled with cold compresses. In 3 cases granulomas of the conjunctiva occurred, the result of imperfect closure of the conjunctiva at the time of the operation; they were removed later. Considerable thickening and swelling often persisted at the operative site for several months. This condition was due to slow absorption of the catgut. No disability or deformity resulted, and in all cases clearing was uneventful.

Transient diplopia was present in fully 75 per cent of cases immediately after operation. In some cases this persisted for two or three days, with gradual merging of the two images and disappearance of symptoms. In 6 cases annoying diplopia of various degrees was encountered. In 3 of these cases the diplopia proved amenable to orthoptic training, with final complete cure. In 2 of the remaining 3 cases some annoyance continued. In these cases the condition gradually improved under intensive orthoptic training, although the response to treatment was slow. In 1 case, however, the diplopia proved most distressing, and no amount of orthoptic training or mental education could remove this annoying condition. Loss in convergence power resulted in the patient's requiring four operations. This loss was due to a bilateral recession of the internal rectus muscles, performed at different operations. The condition was remedied somewhat by a later advancement of one of the internal rectus muscles, but only at a sacrifice of convergence. Various authors have stressed the possibility of this complication. A regrettable loss of convergence often results from (1) too energetic an operative procedure on both internal rectus muscles or (2) too liberal a recession of one internal rectus muscle. Experience has shown me that it is inadvisable either to perform recession of both internal rectus muscles in the same patient or to reinsert a recessed internal rectus muscle posterior to the equator. Thus, a recession of more than 5 mm. always entails the possibility of future loss of convergence power. Any remaining deviation is best treated by a future operation on the external rectus muscles.

SUMMARY AND CONCLUSIONS

In an analysis of 65 cases of strabismus in adults treated by operative procedures, the following conclusions were drawn:

A proper diagnosis and investigation of the condition of the ocular musculature must be made prior to operation. This can be determined by a routine examination. Included in the study should be measurement of the refractive error, a complete history and determination of the presence or absence of fusion.

Recession of one muscle combined with resection and advancement of the opposing muscle is the surgical procedure which offers the best chance for success.

With proper evaluation of the muscle power and appropriate treatment fusion of some degree will be obtained in 60 per cent of cases.

Amblyopia, suppression, poor fixation and anomalous retinal correspondence constitute definite obstacles in the treatment of strabismus.

Diplopia is a complication resulting from the operative treatment of strabismus.

Joseph Sosnowy, technician fourth class, and Private H. Moskowitz assisted in the examination of the patients in this series.

BILATERAL TERATOID TUMOR OF THE LIMBUS

CAPTAIN EMANUEL ROSEN
MEDICAL CORPS, ARMY OF THE UNITED STATES

REPORT OF A CASE

A man aged 25 was first seen at the ophthalmic clinic on Oct. 7, 1943, referred because of poor vision in his right eye. Examination was made to determine the patient's physical fitness for full military duty. The patient stated that as long as he could remember vision had been poor in his right eye, that he had never been aware of any acute symptoms involving this eye and that, although its appearance was somewhat disconcerting, it had caused him neither pain nor concern. His left eye had always been normal, never having produced eyestrain and never having required glasses. The family history was insignificant as regards ocular pathologic conditions; no members of the immediate family had had any ocular tumors or other diseases of the eye. His own past history was of no importance ophthalmologically since he had suffered from no unusual diseases of childhood and had never been hospitalized except for a fracture of the wrist.

Examination revealed vision to be 5/200 in the right eye and 20/20 in the left eye. The lids and the lacrimal apparatus were not remarkable; muscle balance was not unusual except for a remote near point of convergence. There was a peculiar dusky red injection of the bulbar conjunctiva, particularly in the right eye. In this eye three fleshy hypervascularized masses were located just on the limbus—two in the upper part of the bulbar conjunctiva and one in the inferior portion of the globe. When the upper lid was everted and the patient looked to the extreme inferior field, these upper fleshy masses seemed to run well up into the cul-de-sac, the temporal lesion joining the region of the lacrimal gland and the nasal mass continuing into the nasal portion of the cul-de-sac. These masses were grayish, slightly elevated and sharply outlined. They extended beyond the corneal limbus for 2 mm. The vascularization increased centripetally. Each mass was approximately 5 mm. in diameter. The cornea of this eye was irregularly staphylomatous. With the slit lamp the corneal bulge and the decreased corneal thickness were readily apparent. The anterior chamber consequently was extremely deep at certain sections. There was nothing remarkable about the iris or the lens; the fundus was only indistinctly visualized, for no landmarks could be observed. There was no increase of intraocular tension determinable by means of digital palpation (fig. 1).

The left eye showed two smaller masses of similar but less vascular appearance. These masses were located in the upper sector of the globe, with an extension approximately beyond the limbus for 1 mm. There was no scarring of the cornea, no apparent effect on the various radii of curvature and no corneal astigmatism. The remaining ocular structures were all normal.

The general physical examination of the patient revealed nothing of moment. There were no cutaneous tumors and no pigmented nevi or verrucae. The patient had no birthmarks and no congenital anomalies. There were no aberrations of the skeletal, muscular or nervous system or of other special sense organs which were investigated. The heart, lungs and blood pressure were not remarkable.

Because of the presence of these tumors in the eyes and because of their resemblance to "dermoid tumors" of the conjunctiva, although no cilia were visible anywhere on the tumors, it was decided to perform a biopsy of one of these masses for diagnostic purpose.

The temporal mass in the right eye was dissected free, a local anesthetic being used. The mass was readily removed, with only a moderate amount of bleeding. A continuous silk suture, which was removed in five days, was inserted into the conjunctiva for closure of the wound.

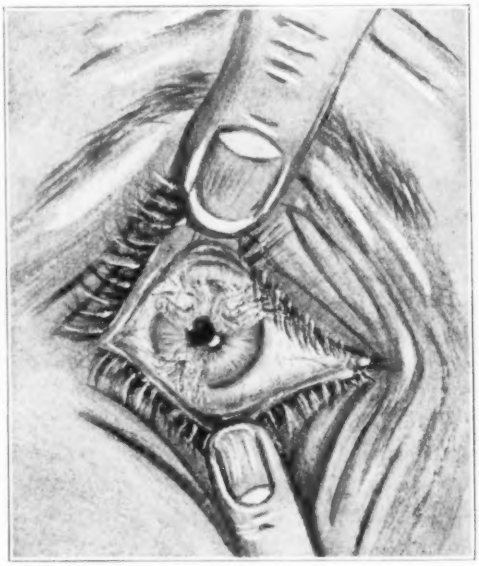

Fig. 1.—Drawing of right eye, showing three fleshy hypervascularized masses on the limbus.

The pathologic report was as follows: macroscopically the specimen consisted of a small piece of pale, soft, noncystic tissue measuring 6 by 5 by 5 mm.

Microscopically the section showed a tissue filled with many loose adipose structures, in which there were many islands of lacrimal gland. The adipose tissue took on the form of hexagons, with small, flattened, densely stained, oval nuclei. There were only a few areas of adipose tissue mixed with tissue of the lacrimal gland proper. There were several areas of adipose tissue intermixed with a loose form of connective tissue. The connective tissue in general was loose and laden with many red blood cells and plasma cells, indicative of its extreme vascularity.

There were three large areas of cartilaginous tissue, located at different positions in the section. Each was surrounded by a richly nuclear perichondrium. These cartilaginous islands were located in a zone of loose connective tissue.

There were many islands of glandular tissue. These included alveolar ducts as well as tissue of the gland proper. Most of the cells were vacuolated with the short oval nuclei crowded toward the base of the gland. Most of these glandular masses were embedded in thin adipose and connective tissue (fig. 2).

In one small area there was an incomplete section of conjunctiva, which consisted of several rows of epithelial cells located on moderately vascular subconjunctival tissue. An occasional bundle of nerve fibers was seen.

Wilson [1] reported 2 cases of teratoid tumor. The first occurred in a baby boy aged 2 months, who was originally examined because of bilateral congenital notched lids. In the right eye there was a large limbal tumor, which covered the lateral third of the cornea and some of the conjunctiva beyond. A similar but smaller tumor was present in the left eye. These masses were hard and closely adherent to the globe. Microscopically they showed hair follicles and sebaceous glands just beneath the surface epithelium. Cartilaginous tissue surrounded by a fibrolipomatous capsule was also present. Wilson's second case was reported as a case of dermolipoma or "teratomatous" tumor. This tumor was oval, yellowish and subconjunctival. It had increased slowly in size and had extended just onto the cornea. The excised specimen "consisted of a mass of fatty tissue partially enclosed in a fibrous capsule. There was an elongated narrow strip of normal hyaline cartilage surrounded by perichondrium." The lesion in Wilson's first case represented a form of limbal dermoid frequently associated with coloboma of the upper lid and as such points out the congenital nature of the abnormality, as distinguished from a new growth.

Teratomatous tumors of the lacrimal gland are occasionally misdiagnosed. They have been called dislocated lacrimal gland; aberrant lacrimal gland with ectopic cartilage; teratoma; mixed lacrimal gland tumor; dermoid; teratoid tumor, and congenital osteoma of the conjunctiva.[2a] A patient with such a tumor may show a defect in the upper lid on the involved side with a peculiar pendulous elongation, which may be pulled down over the eye.[2] In some cases the mass may resemble the not uncommon dermoid of the limbus, but the pathologic section will usually show the true nature of the tumor.

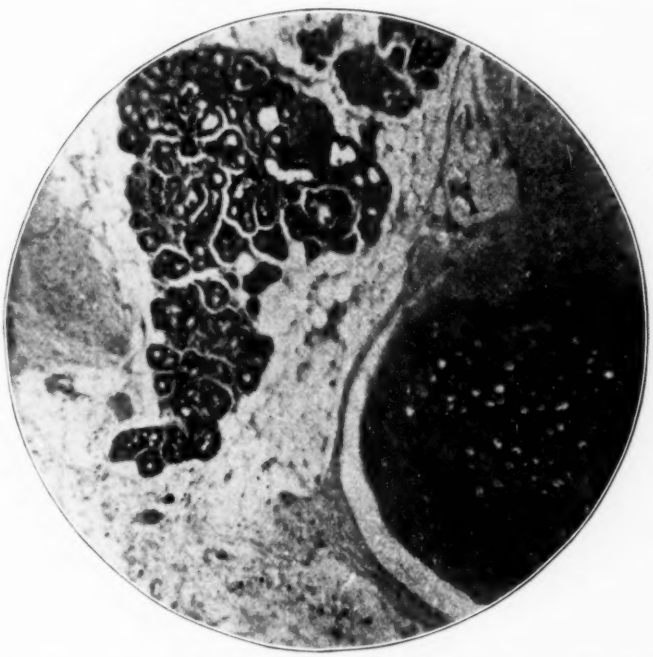

Fig. 2.—Photomicrograph showing area of glandular tissue with cartilage.

1. Bilateral Dermoid Tumor of Limbus with Bilateral Coloboma of the Lid, in Eighth Annual Report of the Giza Memorial Ophthalmic Laboratory (1933), Cairo, Schindler's Press, 1934, p. 54. Dermo-Lipoma of the Conjunctiva Containing Cartilage, in Thirteenth Annual Report of the Giza Memorial Ophthalmic Laboratory (1938), Cairo, Schindler's Press, 1940, p. 54.

2. Bon Hevven, J. A.: Nederl. tijdschr. v. geneesk. **72**:3566, 1928.

2a. Parsons, J. H.: Pathology of the Eye, New York, G. P. Putnam's Sons, 1908. Snell, S.: Ophth. Rev. **1**:207, 1881-1882. Coppez, Arch. d. opht. **3**:203, 1903. Carter, L. F.: Am. J. Ophth. **27**:67, 1944.

The occurrence of such a tumor at the limbus or nearby has been explained embryologically as a possible remnant of primordial tissue. A dermal inclusion has been suggested as a possible explanation of the site and type of this tumor. Areas of fetal clefts and regions where fetal adhesions might persist and produce pressure have been located. It has been shown [3] that ectodermal inclusion along the lines of the closing facial fissures is not the entire causative factor, although it is usually true that these tumors occur opposite the cranial fetal suture lines.

In 1943 Sherman [4] reported a case of "teratoid tumor of conjunctiva and other developmental anomalies with naevus verrucosus of scalp." The excised tissue showed "serous glands surrounded by loose areolar tissue and dilated endothelial spaces resembling lymphatics with cartilage and a few nerves cut obliquely and transversely." It was pointed out in the discussion that such growths should be called teratoid tumors. Duke-Elder [5] showed that the usual site is between the external and superior rectus muscles. The presence of a "phacoma" located in the midline in a position occupied by a fetal cleft and associated with a corneal tumor classified as the second type by Ida Mann [3] lends strong support to the theory advanced by Collins and Mayou [6] that these tumors occur opposite the site of bony sutures.

Stargardt [7] suggested that these tumors are congenital anomalies pathologically listed as monophyletic teratoblastomas, since they develop from a single germ layer.

3. Mann, I.: Developmental Abnormalities of the Eye, London, Cambridge University Press, 1937, p. 379.

4. Sherman, A. R.: Teratoid Tumor of Conjunctiva and Other Developmental Anomalies with Naevus Verrucosus of Scalp, Arch. Ophth. **29**:441 (March) 1943.

5. Duke-Elder, W. S.: Text-Book of Ophthalmology, St. Louis, C. V. Mosby Company, 1937, vol. 2, p. 1411.

6. Collins, E. T., and Mayou, M. S.: Pathology and Bacteriology of the Eye, ed. 2, Philadelphia, P. Blakiston's Son & Co., 1925.

7. Stargardt, K.: Ztschr. f. Augenh. **37**:5, 1917.

INTRINSIC VARIABILITY OF ASTIGMATIC ERRORS

JOSEPH I. PASCAL, M.D.
NEW YORK

The true astigmatic error, as measured in the principal plane of the eye, is not variable in amount, except as all living processes are not absolutely fixed and static. The variability centers on the astigmatic correction which is the astigmatic error in the plane of the lens. Possibly the term "variability of astigmatic correction" would be more appropriate, except that this may be confused with an allied condition.

This variability can best be shown by a few illustrations. Take a patient with 3 D. of hypermetropic astigmatism as corrected with a + 3.00 D. cyl., axis 90. The vertical meridian is emmetropic; the horizontal meridian is 3 D. hypermetropic in the plane of the lens. In the principal plane of the eye, say, 17 mm. behind the lens, the horizontal meridian is 3.16 D. hypermetropic. The true astigmatism, then, which is the difference in refraction between the two principal meridians, is 3.16 D.

When this patient with his correction focuses at a distance of 1 meter from the principal plane of the eye, he is said to accommodate 1 D. As the eye accommodates equally in all meridians, an accommodation of 1.00 D. brings the emmetropic vertical meridian in focus for the object. But the same amount of accommodation in the horizontal meridian leaves this meridian underaccommodated by about 0.11 D. To make up this shortage, the eye needs a + 3.10 cyl., practically a + 3.12 cyl., axis 90.

When this patient focuses at 25 cm. and the eye accommodates 4.00 D. in all meridians, it is in focus for the emmetropic vertical meridian. But in the horizontal meridian an accommodation of 4.00 D. represents an underaccommodation of about 0.42 D., which in the plane of the lens is a deficiency of about 0.44 D. The eye, therefore, needs now a + 3.44 D. cyl., practically a + 3.50 D. cyl. The + 3.00 cylinder which corrects the astigmatism fully for infinity is about 0.5 D. too weak for vision at 25 cm. Thus, a true astigmatism of 3.16 D. requires a + 3.00 D. cyl. for vision at 20 feet (6 meters), a + 3.12 D. cyl. for vision at 1 meter, a + 3.50 D. cyl. for vision at 25 cm. and, of course, other powers for other distances.

This variability becomes even more notable if the astigmatism is combined with a spherical error. A patient who has corrected vision with a + 3.00 D. sph. ⊃ + 3.00 D. cyl., axis 90, has a true astigmatic error in the principal plane of the eye of 3.51 D., not 3.00 D. When he focuses at 1 meter, he needs a + 3.00 D. sph. ⊃ + 3.12 D. cyl., axis 90, and when he focuses at 25 cm., he needs approximately a + 3.00 D. sph. ⊃ + 3.50 D. cyl., axis 90. These modified corrections are based on the fact that the over-all accommodation is set by the less hypermetropic meridian. Should the over-all accommodation be set by the more hypermetropic meridian, then the nearest correction for 25 cm. is a + 2.50 D. sph. ⊃ + 3.50 D. cyl., axis 90. That is, the lens which corrected the eye for infinity is too strong by 0.5 D. in the sphere and too weak by 0.5 D. in the cylinder when the patient is focusing at 25 cm.

Similar variability, though in a sense in the opposite direction, exists in simple and compound myopic astigmatism. An eye corrected with a — 3.00 D. cylinder for infinity has a true astigmatism of 2.86 D. When it is focusing on near objects, if the amount of accommodation used is set by the emmetropic meridian, then at 1 meter the eye needs approximately a — 3.12 D. cylinder, and at 25 cm., approximately a — 3.50 D. cylinder. Here, again, a true astigmatism of 2.86 D. needs a — 3.00 D. cylinder for infinity, a — 3.12 D. cylinder for vision at 1 meter and a — 3.50 D. cylinder for vision at 25 cm. If the amount of accommodation used is set by the myopic meridian, then at 25 cm. the needed correction, instead of being — 3.00 D. cyl., axis 180, is approximately + 0.50 D. sph. ⊃ — 3.50 D. cyl., axis 180.

Similarly, in the case of compound myopic astigmatism, an eye with a correction of — 3.00 D. sph. ⊃ — 3.00 D. cyl. has a true astigmatism of only 2.58 D. When the eye is focusing at 25 cm., the accommodation required in the less myopic meridian is 3.64 D., and that needed in the more myopic meridian is 3.33 D. Whichever meridian determines the over-all accommodation will

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cause an excess or a deficiency in the other meridian of about one-third diopter (0.31 D.). In the plane of correction this increases to about 0.45 D., or practically 0.5 D.

Other things being equal, the eye will tend to accommodate the minimum amount. Therefore, in cases of simple and compound myopic astigmatism the more myopic meridian is likely to set the pace. This produces a deficiency in the less myopic meridian, equalization of which will need the addition of plus power. The result is a lens which differs both in sphere and in cylinder from the formula for distance.

In cases of mixed astigmatism, the variability of the astigmatism for near vision is still more pronounced. The hypermetropic meridian has to accommodate more than normal, while the myopic meridian has to accommodate less than normal for the same distance. The difference between the two meridians, therefore, becomes more pronounced, so that greater changes in the cylinder are required for near vision. Slight concomitant changes in the sphere may also be required.

How important are these variations? Since it is generally accepted that small astigmatic errors are more apt to cause eyestrain than large errors, it is worth noting that the full correction of an astigmatic error for 20 feet (6 meters) leaves a small variable astigmatic error for every distance within 20 feet.

Another feature of astigmatism which may be considered to be intrinsically variable concerns the axis. The variability is due to any or all of the following factors:

1. Cyclophoria. The axis found monocularly, with the eyes dissociated, will be off axis to the extent of the imbalance.

2. The compensatory rotation of the eye when the head is tilted to either shoulder.

3. The usual binocular extorsion when the eyes change from distant to near vision. This outward rotation of the vertical meridians is so common that it has been termed physiologic, comparable to the so-called physiologic exophoria for near vision. The amount of extorsion varies with the nearness of the object and its position with reference to the horizontal plane. It decreases when the subject is looking below the horizontal plane and increases when he is looking above it. It is therefore of special significance for people who have to use their eyes at close range while looking on a horizontal level or above it. This applies to librarians, tradesmen and others who have to consult items on shelves placed on a level with or above their eyes.

The three aforementioned factors may partly neutralize each other, or they may have a cumulative effect.

SUMMARY

An astigmatic eye which is properly corrected for distance develops a variable amount of a new astigmatic error when accommodating for near vision. The amount of this astigmatism, which may be termed induced astigmatism, varies with the amount and kind of the true astigmatism. It also varies with the position of the correcting lens, with the nearness of the object viewed and with whether the amount of accommodation used is determined by the meridian of greatest power or the meridian of least power. The last factor probably depends on the contours of the object looked at. The amount of this induced astigmatism is ordinarily slight but can easily amount to 0.5 D.

A similar variability in the position of the axis is due to the rotation of the eye on an anteroposterior axis, occasioned by three factors: (*a*) cyclophoria, (*b*) a compensatory rotation due to tilting the head and, in near fixation, (*c*) physiologic binocular extorsion. The extent of change in axis is ordinarily low, amounting possibly to 5 or 10 degrees.

There is no way of entirely eliminating the incidence of these intrinsic variabilities. But a few suggestions may be offered.

(*a*) When in the usual monocular tests for astigmatism at 20 feet (6 meters) the location of the accepted axis varies within a range of, say, 5 or 10 degrees, it is preferable to choose the axis in line with the physiologic extorsion, that is, the more temporal position. (*b*) Since it is generally more important to have keen, comfortable vision for near than for distant vision, one may obtain a compromise correction by making the astigmatic test at some intermediate near point, e. g., 40 or 50 cm., changing only the spherical element for the 20 foot test; and (*c*) locating the cylinder axis in each eye during binocular fixation.

Finally, while it is pertinent to know of the existence of induced astigmatism and shifts in axis as occasioned by the factors described, these defects do not in general cause any distress or measurably lessened visual acuity. Possibly the eyes accommodate for the circle of least diffusion; or, more likely, the errors fall within the adaptability of the eye as a living dynamic organ. But for those sensitive patients who are distressed when their correction is off an eighth or a quarter of a diopter, or the cylinder axis is off a fraction of 5 degrees, these considerations should be taken into account.

37 West Ninety-Seventh Street.

FOSTER KENNEDY SYNDROME WITH FUSIFORM ANEURYSM OF INTERNAL CAROTID ARTERIES

I. S. TASSMAN, M.D.
PHILADELPHIA

The location of an aneurysm is the important factor in determining the focal signs. Garvey,[1] in a review of the subject of "Aneurysms of the Circle of Willis," described those aneurysms which arise from the intracranial portion of the carotid artery, from the proximal portions of the middle cerebral artery and from the circle of Willis at the point of bifurcation of these vessels as the anterior group, which presents a remarkably constant clinical picture. Also, because of the higher incidence of aneurysms of the carotid portion of the circle of Willis and the constancy of the clinical picture, the aneurysms in this location are diagnosed more frequently than those elsewhere.

Localized pain in the supraorbital region on the side of the aneurysm may be present for a long time before focal signs appear. Other symptoms resulting from direct pressure on adjacent structures develop, with increase in size of the aneurysm. These may include ptosis, diplopia, intermittent sharp pain over the upper portion of the face and impairment of vision. The oculomotor nerve and less frequently the sensory portion of the trigeminal nerve, the motor division of the trigeminal nerve and the olfactory, optic, trochlear and abducens nerves may be involved by an aneurysm arising from the carotid portion of the circle of Willis. Although compression of the optic nerve on the side of the aneurysm occurs, there may be little evidence of disturbed function.

Many types of defects of the visual field have also been described in cases of such aneurysm. Bitemporal hemianopsia, defects of the nasal field and others have been found.

The case here reported is striking in that it presented a characteristic Foster Kennedy syndrome and, with the exception of pain, almost complete absence of other focal signs and symptoms.

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REPORT OF A CASE

Mrs. S. F., a white woman aged 32, was first seen by me at Wills Hospital on Nov. 17, 1943. She had been referred with a diagnosis of optic neuritis of the right eye and secondary atrophy of the left optic nerve. She was admitted to the hospital on the same day, with the chief complaint of "pain over the right eye and loss of vision in the left eye."

Past History.—The patient is the mother of two children. She suffered no serious illness until July 1942, when she had a pelvic operation, the exact nature of which is not known.

In April 1943 she suffered with herpes zoster, which affected principally the left side and the back of the neck. There was no history of other illnesses or of injuries.

Present Illness.—The patient first complained of pain in the left eye in January 1943. She thought she had suffered with a "cold" just prior to this. Shortly afterward, she noticed failure of vision in the left eye. After consulting an ophthalmologist in Reading, Pa., she had some teeth extracted and her sinuses examined. She was hospitalized there and placed under treatment, which included active fever therapy. Vision in the left eye continued to decrease, however, and in October 1943 she began to have pain in the right eye. She then was told that the right eye was also involved. By November 1943 vision in the left eye was almost entirely gone.

Ocular Examination.—On admission to Wills Hospital, the patient revealed no abnormalities of development or in her general physical makeup. Her head was of normal size and contour. Speech, hearing and smell were not affected.

Vision in the right eye was 6/6 partly; with the left eye she could see hand movements at 12 inches (30 cm.). The eyes were in normal position, and the cilia, the lacrimal apparatus and the conjunctiva were all normal.

The action of the lids was good, and no ptosis or nystagmus was noted. The ocular rotations were full in all directions. The pupils, equal in size, measured about 5 mm. in diameter. They were round and regular in outline and failed to react to direct light. The anterior chambers were normal in depth, and the tension was normal in both eyes.

Ophthalmoscopic examination revealed the media to be clear in each eye. The right eye showed a definite papilledema measuring about 3 diopters, with marked blurring of the margins of the disk. The retinal veins were enlarged and tortuous, and the arteries were slightly increased in size, but none of the vessels showed any evidence of the presence of an inflammatory process. No hemorrhages, exudates or other pathologic conditions were found in the periphery of the fundus.

In the left eye, the margins of the disk were slightly blurred; the disk was white, with a deep excavation in the center. The retinal vessels showed no evidence of inflammation, and there were no hemorrhages or other pathologic conditions in the periphery. The macula was normal.

The presence of definite papilledema in the right eye with good visual acuity and atrophy of the optic nerve in the left eye with loss of vision suggested a Foster Kennedy syndrome with an intracranial lesion.

Visual Fields: Examination of the right eye revealed a moderate contraction of the peripheral field and a slight enlargement of the blindspot. There was insufficient vision in the left eye for a test of the visual fields.

Examination of the Ears, Nose and Throat.—Conditions were normal except for the presence of small and submerged tonsils.

Roentgenographic Examination of the Skull and Sinuses (Dr. Fetter).—The sella turcica appeared normal and oval, and there was slight calcification in the petrous ligaments.

The bones of the skull were a little thicker than average, but their density was about usual. There was no roentgenographic evidence of increased intracranial pressure, calcification or visible outline of a tumor. The mastoids were moderately developed and had a mixed type of cells, some of which showed considerable thickening of cellular elements.

It was concluded that the skull and the sella turcica showed no evidence of pathologic condition, that mucosal hypertrophy and some exudate could be present in the right ethmoid bones and that slight mucosal hypertrophy existed in the right antrum. The other sinuses were clear.

Laboratory Studies.—Urinalysis revealed that the urine had an acid reaction, a specific gravity of 1.029. It contained no sugar, but there was sufficient albumin to give it a slightly cloudy appearance. A microscopic examination of the urine showed 25 white blood cells per high power field, many red blood cells, several squamous epithelial cells and amorphous urates.

The blood had a sugar content of 104 mg. per hundred cubic centimeters and gave an anticomplementary reaction to the Wassermann test and a negative reaction to the Kahn test.

Examination of the spinal fluid showed a pressure of 110 mm. of water, no cells and no globulin and a total protein content of 25 mg. per hundred cubic centimeters. The results of a colloidal gold test and Kahn and Wassermann tests were negative.

A Mantoux test gave negative results.

Neurologic Examination.—The patient was examined by both Dr. N. Schlezinger and Dr. Bernard Alpers. They suggested the possibility of a suprasellar lesion, either neoplasm or aneurysm, with a Foster Kennedy syndrome. Other localizing neurologic signs were absent.

Dr. Alpers' Report: "I believe this patient has a Foster Kennedy syndrome. The appearance of the right disk seems to me to be that of edema rather than of neuritis. The normal visual acuity in the right eye favors edema rather than neuritis in my estimation. I think the most likely diagnosis is a lesion above the sella turcica, probably compressing the optic nerve and the chiasm. A tumor seems most probable, but multiple sclerosis cannot be excluded. Herpes encephalitis is a possibility, but I am unaware of an encephalitis of herpetic origin secondary to cervical herpes."

In accordance with the recommendation of the neurologist, the patient was transferred to Jefferson Medical College Hospital on Nov. 30, 1943, for further studies and neurosurgical treatment.

Summary of Report of Neurosurgeon, Dr. Rudolph Jaeger, Jefferson Hospital: On Dec. 2, 1943 encephalography was performed. Routine roentgen study of the skull indicated a normal condition. Roentgen study following encephalography showed no evidence of dilatation of any of the ventricles. The lateral ventricles were symmetric and in the midline. However, there appeared to be a definite lack of sharpness in the extreme anterior portion of the lateral ventricles, which raised the question of possible encroachment on the anterior horn by a suspected suprasellar lesion. In addition, the cisterna chiasmatica, while outlined, was not as large as frequently seen, and this may also have been the result of a mass lesion in this area. The rest of the basal cisterna and the subarachnoid pathways appeared normal.

Interpretation: In view of the patient's clinical history, the lack of good filling of the extreme anterior horn and the small size of the cisterna chiasmatica raised the question of a large suprasellar lesion (Teplick).

Reports on Visual Fields (Dec. 2, 1943).—No field was obtained with the left eye. In the right eye there were slight concentric constriction and slight enlargement of the blindspot, with visual acuity of 20/30.

Report of Operative Procedures.—Craniotomy (Dec. 14, 1943): A flap of bone was turned down over the left frontal lobe. There were no adhesions about the optic nerve or strictures around the chiasm. The left nerve was of normal size and appearance. It was entirely free and not compressed by any visible lesion. Just beneath it was a fusiform aneurysm of the internal carotid artery, which after emerging from the base of the skull formed a complete loop into the region of the sella turcica and then passed backward to its normal location to progress laterally to the middle cerebral artery. It appeared as though the artery was bound to the base of the skull by the ophthalmic artery. On the right side there was a similar fusiform aneurysm, with a mass of tiny veins on it, just beneath the optic nerve. This pressed the optic nerve backward tightly against the posterior edge of the optic foramen. It was impossible to do anything surgically with the lesion. It is believed that ocular difficulty on the left side was caused by sclerosis of the ophthalmic artery, because obviously there was not enough pressure against the optic nerve to cause atrophy and blindness. The pressure against the optic nerve on the right side was definite enough to cause progressive blindness, although one would think there would be atrophy rather than edema of the disk on that side. The pressure, however, may have been just sufficient to cause congestion of the venous blood returning from the orbit. The roof of the orbit over the top of the optic nerve should have been removed to relieve the pressure against it, but it was impossible to do this through the left frontal lobe. This should be kept in mind so that the procedure may be carried out by an operation through the right frontal lobe, should blindness set in and become progressive. The whole chiasm and both optic nerves were completely exposed without difficulty. The bone flap was wired in place after the dura was tightly closed. The skin was sutured with silk, with a Penrose drain beneath the flap.

Postoperative Diagnosis: The diagnosis was bilateral fusiform aneurysm of the internal carotid artery.

Report on Visual Fields (Jan. 6, 1944): No field was obtained with the left eye. There was marked concentric constriction in the right eye. Visual acuity was 20/70. Arteriography (Jan. 14, 1944): The right carotid arteries were exposed. The external carotid artery was temporarily occluded. Fifteen cubic centimeters of 35 per cent diodrast solution was injected rapidly into the internal carotid artery. Roentgenograms taken after this showed a tortuous internal carotid artery just beneath the right optic nerve, as observed at a previous operation. An aluminum clip was loosely placed around the internal carotid artery, in order to occlude this vessel later, if it seemed to be advisable.

Postoperative Diagnosis: The diagnosis was fusiform intracranial aneurysm of the internal carotid artery on the right side.

Craniotomy (Jan. 19, 1944). A flap of bone was turned down over the right frontal lobe. The right optic nerve was exposed without difficulty. The internal carotid artery fitted snuggly beneath it and pressed it rather tightly against the upper edge of the optic foramen. A part of the vessel could be seen to the left of the optic nerve. The roof of the optic canal was removed for a distance of about ½ inch (1.27 cm.) from the foramen. The left optic nerve was visualized and appeared to be more snuggly pressed against the edge of the foramen than was obvious at the first operation. The right optic nerve appeared to be of good bulk and was not as tightly constricted as it should be to produce the loss of vision which occurred. Apparently the visual loss had to do at least partly with the papilledema caused by venous congestion. The dura was sutured tightly. The bone flap was wired in place and the muscles and skin closed with silk.

Postoperative Diagnosis: The diagnosis was fusiform aneurysm of the carotid artery.

Further Course (Jan. 24, 1944): The aluminum clip which had been placed about the right internal carotid artery was removed. It appeared to have completely occluded the internal carotid artery, owing to adhesions, although the clip was by no means tightly closed. The wound was sutured with silk.

Report of Visual Fields (Jan. 29, 1944): No field was obtained with the left eye. In the right eye there was further constriction of the visual field. Visual acuity was 5/200.

SUMMARY

A married woman aged 32 suffered with pain over the eyes and loss of vision in the left eye over about eight months. Other subjective signs and complaints were practically absent. Ocular examination revealed the presence of a Foster Kennedy syndrome (papilledema with good vision in the right eye and atrophy of the optic nerve and almost total loss of vision in the left eye). The presence of a suprasellar lesion was suspected. Definite diagnosis was made at the time of operation, which revealed bilateral fusiform aneurysms of the internal carotid arteries, with formation of anomalous loops in the course of the arteries and compression of the optic nerves.

CONCLUSION

This case was considered to be of especial interest to the ophthalmologist from the standpoint of the ocular involvement that was revealed and the lack of other major signs and symptoms.

Since it was first described by Foster Kennedy,[2] or perhaps even earlier by Paton,[3] the syndrome has been considered to indicate the presence of a basofrontal tumor. However, a number of cases have been reported since that time in which it was present with non-neoplastic conditions. One of these was a case of aneurysm of the right internal carotid artery later reported by Kennedy himself. Most of the others were cases of arteriosclerosis or atherosclerosis of the vessels with compression of the optic nerves. H. E. Yaskin and N. S. Schlezinger [4] described 2 such cases and referred to about 11 others which they found in the literature associated with various other conditions. Among these was 1 (Cusick) which was reported in association with a tumor of the cerebellum. The ocular changes in this case were said to be caused by extreme dilatation and forward displacement of the third ventricle.

Because of the anomalous vascular condition found at the time of operation, it is felt that the case here reported is one of congenital origin.

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PATHOGENESIS OF ACUTE GLAUCOMA

LEO HESS, M.D.
BOSTON

"Like the eyes are not to be treated without the skull and the skull is not to be treated without the entire body, the body is not to be treated without the soul. The Greek physicians fail to cure many diseases because they ignore the entire entity of man. If the entire body does not feel good, a part of it cannot be improved."
—Plato, Charmides 156 E.

It is generally agreed that the clinical signs of acute "inflammatory" glaucoma are caused by increased intraocular pressure (Fuchs [1]). Treatment is directed, in accordance with this classic teaching, toward lowering the tension, in order to avoid damage to the tissues of the eyeball, particularly to the optic nerve. The cause and the mechanism of the increased tension are still unexplained. In an earlier paper [2] I expressed the view that the pathologic hardening of the eyeball with glaucoma is analogous to the physiologic orgasm, both events being dependent on reactions within the vegetative nervous system. The integrated clinical picture, with its numerous signs, viewed broadly may be regarded as a vegetative neurovascular crisis, other examples of which are epileptic seizures (particularly the type occurring during digestion, which can be described as a "sympathetic crisis") and attacks of cardiac asthma associated with acute edema of the lungs (Hess [3]).

An outstanding clinical sign of acute glaucoma is venous congestion of most of the tissues of the eye. Dilated venous loops in the center of the optic disk are not uncommon. A "head of Medusa" may occasionally be observed in the bulbar conjunctiva. The veins of the orbit communicate with many venous sinuses and plexuses nearby, all richly endowed with nervous plexuses and ganglion cells. These elements are presumably important for the regulation of the venous blood stream within the bulbus. The arterial branches of the eyeground are frequently narrowed and the delicate temporal rami may altogether disappear. This narrowing of the arterioles cannot be adequately accounted for by increased intraocular pressure. It is more likely that it is caused by increased activity of certain vasomotor nerves. The immediate effect of arteriolar constriction and partial ischemia is dilatation of the capillaries and later of the smallest veins (Krogh [4]). The rise in intracapillary pressure and the dilatation of the capillaries and of the nearby venules is necessarily associated with the escape of plasma by filtration, with increase of the fluid content of the bulbus. It appears that the relationship between the increased intraocular pressure, on the one hand, and the vascular changes, the increased transudation of fluid and finally the so-called glaucomatous atrophy of the optic nerve, on the other hand, is not as simple as some investigators are inclined to suppose.

SIGNS OF ACUTE GLAUCOMA

It is necessary to analyze the main signs and symptoms of acute glaucoma briefly in order to establish a theory of pathogenesis which can account for the entire clinical picture on a rational basis.

Hardening of the Eyeball.—Hardening of the eyeball in glaucoma is caused by an increase in the content of fluid with resulting occlusion of the angles of the chamber and congestion of the choroid, sclera and conjunctiva. The increase in the content of fluid may be the result of increase in the arterial blood supply, of impairment of the venous return or of both. In addition to these factors, the increase in capillary transudation is to be borne in mind. In physiologic hardening, as exemplified by the orgasm, both factors operate without resulting transudation. Since there is no evidence of arterial hyperemia in glaucoma, venous congestion remains for consideration. The occurrence of the prodromes and of the attacks after hearty meals, after emotion and in association with objective signs of heart failure suggests that venous congestion plays a precipitating role.

Dr. Hess was formerly Professor of Medicine and Neurology at the University of Vienna.

Dr. Boruchoff and Dr. Elsberg, of the Eye Clinic of Beth Israel Hospital, gave assistance in this study.

1. Fuchs, E.: Lehrbuch der Augenheilkunde, Leipzig, Franz Deuticke, 1939, p. 426.

2. Hess, L.: Pathology of Acute Glaucoma, Arch. Ophth. **26**:250 (Aug.) 1941.

3. Hess, L.: Deutsches Arch. f. klin. Med. **173**: 283, 1932.

4. Krogh, A.: The Anatomy and Physiology of Capillaries, ed. 2, New Haven, Conn., Yale University Press, 1929.

However, it cannot be denied that a primary, transient rise in the blood pressure, for example, after nervous strain, may antedate the venous congestion.

The bearing of venous congestion on the causation of glaucoma is illustrated by the following report on a case.

H. A., a 41 year old housewife, had nosebleed for a week. The bleeding was stopped by cauterization. Some years previously she had had hematometra as a result of constriction of the cervix of the uterus. Menstrual bleeding was irregular and occasionally prolonged. She had pains in the right flank and hematuria. Compensated heart disease was present. The patient was adipose, with a flushed "full moon" face, and the veins on both sides of the neck were slightly distended.[5] The veins of the conjunctiva were tortuous and congested, especially in the left eye, and the retinal veins were lightly tortuous. Small points and streaks appeared in the lens. There was no cupping of the optic disk, and the peripheral fields were normal. The intraocular pressure was 56 mm. (Schiøtz) in the right eye and 75 mm. in the left.

Consistent with the hypothesis of venous congestion as a cause of glaucoma are the well known conditions observed in ophthalmoscopic examination after an attack. Simultaneous arterial narrowing and venous distention can be explained satisfactorily as a neurovascular phenomenon.[6] Primary constriction of the arterioles may give rise to reflex dilatation of capillaries and veins (Krogh[4]), along with increased transudation of fluid. Glaucoma due to thrombosis of small branches of the ophthalmic vein, with secondary collateral dilatation of other veins and increased intraocular pressure was described by Magitot and Bailliart.[7]

Mydriasis.—Dilatation of the pupil is a common and impressive sign before and during attacks of glaucoma. At first it is transitory, but it may last for several days and in the advanced stages of the disease may become permanent. Similar persistent mydriasis may be elicited by instillation of atropine in the eyes of patients with sympathicotonia.[8] Not only is the pupil of the involved eye much larger than the other pupil, but it is sometimes oval rather than round, and it may be eccentric. The dilated, oval pupil does not react to light or accommodate for distance. These findings suggest a hyperactive or spastic condition of the dilator muscle of the iris, which is innervated by sympathetic fibers. In the early stages this hyperactivity must be functional, because it is transitory; later it is persistent and must be caused by an organic lesion.

Sympathetic stimulation of the dilator muscle of the iris may arise in the diencephalon, in spinal centers or in peripheral sympathetic fibers or ganglions. Karplus and Kreidl[9] described a vegetative center in the diencephalon not far behind the optic tracts and lateral to the infundibulum. Stimulation of this area in animals results in maximum dilatation of the pupils, widening of the eye slits, retraction of the so-called inner lid, lacrimation and vasomotor reactions. Budge[10] described the ciliospinal sympathetic center, at the level of the eighth cervical and first and second thoracic segments of the spinal cord. From the ciliospinal center arise certain fibers which reach the superior cervical ganglion and pass by way of the carotid plexus to the gasserian ganglion. From there they course via the ophthalmic branch of the trigeminal nerve and the long ciliary nerves to the vessels of the eyeball, the dilator muscle of the iris and the müllerian muscle. These are the so-called peripheral sympathetic fibers of the eye.

In which part of the sympathetic system may the stimulus producing glaucoma arise?

There is no reason to suppose a mechanical irritation of the peripheral sympathetic fibers or ganglions such as may be produced by enlargement of the cervical or mediastinal lymph nodes or of the thyroid gland. Nor is there evidence of chemical stimulation, such as is produced by excessive secretion of epinephrine, as occurs with sympathoblastoma. Functional hyperactivity of the peripheral portion of the sympathetic nervous system would involve, according to the concept of Eppinger and Hess,[8] the entire system. But in glaucoma the nervous disturbance is limited to the eyeball and the surrounding structures (conjunctiva and lids), the inner organs apparently being exempt.

A central origin, either within the spinal cord or within the brain, must be considered. Mydriasis due to irritation of the ciliospinal center is rare except as a result of injury at birth, and it is associated with involvement of the cervical or the thoracic portion of the spinal cord, which is absent in glaucoma. Accordingly, a cerebral factor must be considered, perhaps

5. The venous congestion reminds one of the "plethoric" facies seen in patients with Cushing's syndrome (adrenal cortical hyperfunction).

6. As early as 1846 Tavignot suggested a nervous disorder of the ciliary body as a cause of glaucoma.

7. Magitot, A., and Bailliart, P.: Ann. d'ocul. **162**: 729, 1925; Am. J. Ophth. **8**:761, 1925.

8. Eppinger, H., and Hess, L.: Ztschr. f. klin. Med. **68**:205 and 230, 1909.

9. Karplus, I. P., and Kreidl, A.: Arch. f. d. ges. Physiol. **129**:138, 1909; **135**:401, 1910; **143**:109, 1911; **171**:192, 1918; **215**:667, 1927.

10. Budge, J.: Ueber die Bewegungen der Iris, Braunschweig, Fr. Vieweg u. Sohn, 1855, chap. 4.

involvement of the center described by Karplus and Kreidl. The clinical findings are consistent with the effects of stimulation of this center.

In addition to mydriasis, which is absent only rarely, a slight bulging of the bulbus may sometimes be noticed in patients with glaucoma. The widening of the interpalpebral fissure is probably masked by the acute swelling of the lids and the conjunctivas.

The sympathetic nervous system produces "crises" analogous to that of glaucoma, as has already been suggested.

In this relation I should like to cite a case reported by Wallenberg.[11] The patient had hypotonia, atrophy and chorea of the muscles of the right arm and leg, anhidrosis of the right half of the body and mydriasis and enlargement of the eye slit of the right eye. The organic lesion was in the right side of the base of the brain close to the right corpus subthalamicum, that is, in the vicinity of the Karplus-Kreidl center. The oculopupillary syndrome resulting from the diencephalic lesion is remarkable evidence in support of the assumption that mydriasis associated with glaucoma is of cerebral origin.

The condition known as "hemicrania sympathica" is a phenomenon related to glaucoma, characterized by pallor of one half of the face, cordlike contraction of the temporal artery (it is "like pieces of wire" according to Lauder Brunton), dilatation and fixity of the homolateral pupil, production of a small amount of tenacious saliva ("sympathetic saliva") according to Ludwig), high pulse rate, one-sided headache and vomiting. The veins of the retina may be engorged and its small arteries constricted. The similarity of this condition, which is generally agreed to be dependent on increased sympathetic influences, to acute glaucoma, except for the changes within the eye, is obvious.

Epileptic seizures likewise bear numerous signs of sympathetic crises (fixed, large pupils, general pallor, sometimes failure of the peripheral pulse at the beginning of the attack and constriction of the retinal arteries). The origin of the sympathetic hyperactivity in migraine and in epilepsy needs further investigation. Suffice it to say that all of these conditions are the result of sympathetic stimulation. If it is assumed that the primary site of the lesion in acute glaucoma is within the diencephalon, all the main signs must be logically accounted for on this basis. In addition to the diencephalon, the ciliary ganglion, as an important reflex center of the eyeball, deserves full attention.[2]

Headache.—Patients with glaucoma complain of frontal headache or of pain in one temple, radiating to the upper jaw or to one ear. The type of headache is that of intracranial neuralgia of the trigeminal nerve, simulating the pain of migraine. Central stimulation of the Karplus-Kreidl center could easily result in referred pain in this region. The stimulus may spread from there through the peduncles to the cervical portion of the spinal cord, to the ciliospinal center (Budge[10]) and finally, through the rami communicantes, to the sympathetic trunk. It may be conveyed by sympathetic fibers, arising from the first cervical ganglion of the sympathetic trunk, to the gasserian ganglion. The unilateral headache with nausea and vomiting remind one of migraine, especially as many patients with glaucoma, like those with migraine, keep their eyes closed and prefer a dark room. Headache, nausea and vomiting may persist for one or two weeks after the attack, and the clinical picture reminds one of gastric crisis due to tabes dorsalis, the nervous origin of which is not doubtful. Nausea may be the premonitory sign of a new attack of glaucoma.

Hyperthermia.—Since there is no infection or demonstrable toxic agent, it may be inferred that the hyperthermia that occasionally accompanies acute glaucoma is of central origin. An important heat-regulating center is located in the area of the tuber cinereum, which is close to the Karplus-Kreidl center. This heat-regulating center is made up of a sympathetic and a parasympathetic portion. Sympathomimetic drugs (tetrahydronaphthylamine, epinephrine, ephedrine, caffeine and cocaine) cause a rise of temperature by specific stimulation of certain cells within the heat center, and the parasympathomimetic drugs (pilocarpine, picrotoxin, veratrine and santonin) are able to reduce the temperature of the body through their specific action on other cells in the same center. High temperature may be encountered without any infection as a result of central stimulation in: heat prostration; cerebral hemorrhage close to the third ventricle, exerting mechanical pressure on the tissue below the third ventricle, including the tuber cinereum; hydrocephalus; injury to the brain, and epilepsy. The Karplus-Kreidl center is likewise believed to be built up of two antagonistic portions. High temperature with glaucoma could therefore result from the same mechanism that operates in the production of fever with injury of the brain, cerebral hemorrhage, hydrocephalus and epilepsy.

11. Wallenberg, H., cited by Müller, L. R.: Lebensnerven und Lebenstriebe, Berlin, Julius Springer, 1931, p. 181.

Photophobia.—Photophobia is a reflex phenomenon arising from irritation of the cornea, conjunctiva or iris, mediated by the trigeminal nerve. It represents hypersensitivity of the retina, which Claude Bernard[12] demonstrated even in blind men and in animals in which the optic nerve was severed. The sensitivity of the retina is decreased by instillation of parasympathomimetic drugs. The sedative action of cocaine is dependent on its action on the terminations of the trigeminal nerve, abolishing the sensory part of the reflex arc.

Chemosis.—Chemosis and swelling of the eyelids might be signs of inflammation, but their similarity to angioneurotic edema and to acute pulmonary edema accompanying organic lesions of the brain (Hess)[13] leads us to suggest a neural origin. With inflammatory conditions of the eye, for example, iritis, the intraocular pressure is lowered.

Lacrimation.—Lacrimation during and sometimes following acute glaucoma is analogous to the profuse lacrimation noticed by Karplus and Kreidl on stimulation of the diencephalic center.

INDIRECT EVIDENCE OF THE INTERVENTION OF A NERVOUS FACTOR IN THE PATHOGENESIS OF ACUTE GLAUCOMA

Periodicity.—Glaucoma exhibits a periodicity resembling that seen in attacks of migraine, cardiac asthma, epilepsy and the attacks of pain and vomiting of gastric crises due to tabes dorsalis.

Bilateral Involvement.—Bilateral involvement, even though the intervening interval may be long, indicates not a local, but a central, nervous disturbance.

Association with Emotional Disturbances.—Undoubtedly there is a relationship between glaucomatous attacks and emotional disturbances (anxiety, fear or worry). Sometimes glaucoma develops in one eye after an operation on the other. Indeed, under conditions of anxiety, increase in the size of the pupils, enlargement of the interpalpebral fissure, slight bulging of the eyes and spasm of the retinal arteries, signs similar to those of acute glaucoma, may be observed.

Relation to Sleep.—According to some investigators the influence of the parasympathetic nerves prevails during sleep. Sleep would therefore represent a natural balance against the hyperactivity of the antagonistic sympathetic nerves. With glaucoma sleep is often disturbed, but the relief of pain, chiefly of the glaucomatous headache, that occurs rarely during sleep reminds one of the similar relief of migraine. It is not without significance to recall the deep sleep that follows the sympathetic crisis of an epileptic seizure.

Trophic Changes.—The neural origin of the so-called "trophic" changes is beyond any doubt, although the mechanism is still obscure, despite the important work by Parker.[14]

Spontaneous Recovery.—Spontaneous recovery, particularly from the prodromal symptoms, is not unusual. Oddly, it is accompanied with a drop in the intraocular pressure to a point below the normal level (to about 15 Schiøtz units). The rise and fall of the tension cannot be accounted for except by a nervous mechanism.

Association with Ophthalmic Herpes Zoster.—Herpes zoster ophthalmicus may antedate an attack of acute glaucoma. This condition is accompanied with unilateral neuralgia referred to the eye, eyebrow and forehead, edema around the eye and sometimes with mydriasis. The neuralgia is apparently intracranial; its primary site is believed to be in the gasserian ganglion.

In one of my patients an inflammatory condition of the cornea was followed by glaucoma.

Bilateral Extension of Symptoms.—The intraocular pressure of the normal eye may rise to some degree during an attack of glaucoma.

Relation to Fever.—Febrile disease of various sorts may precipitate attacks of glaucoma. The relationship is hard to explain satisfactorily, but frequently during febrile episodes the equilibrium of the vegetative nerves is disturbed, with the sympathetic influence prevailing.

PHARMACOLOGIC ASPECT

It is hardly necessary to note that if atropine sulfate is instilled into the fornix of a glaucomatous eye an acute attack may ensue; presumably the atropine inhibits the action of acetylcholine and permits sympathetic dominance. The action of atropine lasts much longer in a glaucomatous than in a normal eye, owing to the fact that the hypoactivity of the parasympathetic nervous system can more easily be overcome. For contraction of the pupil of an untreated glaucomatous eye or after the use of atropine much stronger solutions of pilocarpine, physiostigmine and mecholyl are required, which demonstrates that the parasympathetic tone is low as compared with the normal and that stronger stimulation is needed.

12. Bernard, C.: Leçons sur la physiologie et la pathologie du système nerveux, Paris, J.-B. Baillière, 1858, vol. 2, p. 90.

13. Hess, L.: Wien. med. Wchnschr. **84**:285, 1934.

14. Parker, G. H.: Humoral Agents in Nervous Activity, New York, The Macmillan Company, 1932.

In one of my patients the pupil, contracted by pilocarpine, expanded suddenly during the night and no longer responded to pilocarpine. The change occurred during the first day of profuse metrorrhagia. Instillation of procaine hydrochloride may produce dilatation of the pupil and increase in intraocular pressure. In some cases hypersensitivity to procaine is striking. In some instances of acute glaucoma, after instillation of epinephrine hydrochloride (1:1,000 solution) the pupil becomes dilated. This reaction—the so-called Löwi phenomenon—is evidence of unblocked sympathetic hyperactivity (Hess [15]).

After administration of caffeine by mouth (a cup of black coffee), the pressure in a glaucomatous eye may rise above the normal level; this facilitates early diagnosis, since a normal eye would not show any change. The rise in pressure is evidence of increased irritability of the nerves. The action of caffeine is complicated. It acts on the heat center at the base of the brain (Barbour and Wing [16]), on the vasomotor center and on the muscular walls of the arteries of the brain (Roy and Sherrington [17]).

A histamine-like substance was found by Friedenwald [18] and by Raycroft [19] in the aqueous humor of glaucomatous patients. The same finding has been recorded by Kirwan [20] for patients with epidemic dropsy, characterized by high temperature, diarrhea, anemia, edema of the legs and acute glaucoma.

15. Hess, L.: Epinephrine Mydriasis, Arch. Ophth. **30**:194 (Aug.) 1943.

16. Barbour, H. G., and Wing, E. S.: J. Pharmacol. & Exper. Therap. **5**:105, 1913.

17. Roy, C. S., and Sherrington, C. S.: J. Physiol. **11**:85, 1890.

18, Friedenwald, J. S.: Pathogenesis of Acute Glaucoma, Arch. Ophth. **3**:560 (May) 1930.

19. Raycroft, B. W.: Brit. J. Ophth. **18**:149, 1934.

20. Kirwan, cited by Magitot and Bailliart.[7]

At first glance it is surprising that in some patients with glaucoma the tension is greatly reduced after instillation of pilocarpine or physiostigmine, whereas in normal eyes the tension remains unaltered (Traquair [21]). In these patients apparently the primary sympathicotonic condition produced, by means of a reflex, secondary hypertonia of the antagonist. These signs of counterbalance are not too rare and are confusing to the inexperienced. A similarly "paradoxic" reaction is the absence of mydriasis in rare cases of glaucoma.

Miosis of the pupils, seen with syphilitic conditions of the spinal cord, is, according to my observations, not seldom associated with extreme slowness of response to atropine, probably as a result of hyperactivity of the parasympathetic fibers. This phenomenon is the reverse of the long-lasting response to atropine in glaucomatous eyes.

It is noteworthy that after an attack of glaucoma the response to miotic drugs is more pronounced than during the attack.

CONCLUSIONS

1. Acute glaucoma is a neurovascular crisis.

2. The vegetative diencephalic center at the base of the brain (the Karplus-Kreidl center) and the ciliary ganglion play predominant roles in the nervous mechanism of glaucoma.

3. Increased intraocular pressure and many signs of acute glaucoma are secondary to the central irritation.

4. The so-called inflammatory signs of the attack (dusky redness of the conjunctiva, chemosis, edema of the lids and changes in the cornea) are of angioneurotic origin.

15 Kenwood Street, Brookline, Mass.

21. Traquair, H. M.: Brit. M. J. **2**:933, 1935.

Clinical Notes

EFFECT OF DIETHYLSTILBESTROL ON ACCOMMODATION

LEO L. MAYER, M.D., ST. LOUIS

Clinical research and practical observation at times may call the attention of members of the medical profession to deviations from the normal without providing an insight into the basic physiologic and pathologic changes. This report serves such a function. It lacks a bibliography, a history of the use of diethylstilbestrol or a discussion of the phenomenon of accommodation. Since I have seen accommodative changes in cases in which diethylstilbestrol has been used for several years and have found no previous report of such a case in the literature, it seemed wise to record representative cases for the enlightenment of ophthalmologists and endocrinologists.

Since the advent of the use of diethylstilbestrol or similar androgens for the relief of symptoms of sexual dysfunction in females, or more usually of disturbances of the menopause, I have examined about 40 women with complaints which, when analyzed, were found to be due to difficulties in accommodation. Of this number, I report here the cases of 5, which are representative with respect to symptoms and treatment of five types under which the majority of such cases would fall.

It is well known that presbyopia and the menopause occur in the same or in the preceding or the succeeding decade. My impression when I first began to see patients with difficulties in accommodation was that of precocious presbyopia or premature menopause or both. Gradually, I realized that most women with menopausal symptoms were "taking shots" or were receiving glandular preparations by mouth, and finally I took persistent steps to assure myself, in spite of the patient's answer to the contrary, that she was receiving endocrine therapy, "which could have little to do with the eyes." Such an attitude is characteristic of most patients, as well as of some physicians, who feel that the eyes constitute a separate and distinct portion of the body, unrelated to any other bodily mechanism. It soon became apparent that such reactions were found not only in women with beginning presbyopia but in those who had worn reading glasses or bifocal lenses for several years. Of course the outstanding complaint of the majority of patients was difficulty of near vision. Ordinarily, the answer to this is the prescription of reading glasses, or a change in the strength of the glasses the patient is already wearing. However, when I, or some other ophthalmologist, had changed the lenses three or six months before, further investigation was indicated.

During the past four or five years I have inquired of many ophthalmologists whether they had encountered such cases in their experience and have never had a positive answer. However, during the past year I have had letters and personal communications from physicians who have kept my question in mind and wished to tell me of their experience.

REPORT OF CASES

CASE 1.—Mrs. L. F., aged 38, was emmetropic on examination six years before. Recently she had had difficulty in reading telephone numbers. The past and present histories were without significance except for painful menstruation, for which she had been "taking shots" during the previous month. Examination showed that vision for distance was normal in each eye. Ophthalmologic examination revealed nothing abnormal except that the near point for accommodation was 10 inches (25 cm.). As she appeared too young for presbyopia, I consulted her gynecologist, who agreed to discontinue the injections of diethylstilbestrol. In one week her accommodation returned to normal, the near point being 6 inches (15.2 cm.). Examination six months later showed no change. The painful menstruation was being treated with antispasmodics.

CASE 2.—Miss G. Y., aged 43, had never worn glasses. During the week prior to examination she had noticed a blur before the right eye. Visual acuity was 20/20 in the right eye and 20/30 in the left eye. There was no central scotoma. The visual fields were normal. The fundi were normal. Refraction gave a correction of +0.5 D. sph. for the right eye and of +0.75 D. sph. ⊂ — 0.50 D. cyl., axis 90 for the left eye. The near point of accommodation for the right eye was 9 inches (22.9 cm.), and that for the left eye, 7 inches (17.7 cm.). The patient was given daily doses of 1½ grains (0.097 Gm.) of diethylstilbestrol, and 1 grain (0.065 Gm.) of thyroid, the latter for mild "flashes." The administration of diethylstilbestrol was discontinued, and in two weeks the near point of accommodation for either eye, and for the two together, was 7 inches (17.7 cm.).

CASE 3.—Mrs. G. H., aged 53, had had difficulty with near vision for two weeks. I had her glasses changed four months before and had changed the prescription two years prior to that correction. She had received injections of diethylstilbestrol biweekly during the past year. The additional correction for accommodation two years ago was a +2.75 D. sphere. At the last refraction she accepted a +3.75 D. sphere to bring the near point to 7 inches (20.4 cm.). Injections were reduced to one a month. The patient had a blood pres-

sure of 220 systolic and 140 diastolic. She did not feel as well after the injections were reduced and wanted them increased. After two months' trial the new reading correction, which was rechecked, was accepted.

CASE 4.—Miss P. P., aged 16, had difficulty in focusing her eyes while reading. She had severe dysmenorrhea. She had received increasing doses of diethylstilbestrol by injection during the preceding six months, with some relief of the menstrual condition. Visual acuity for distance was normal in each eye. There was bilateral weakness of accommodation. The near point of accommodation was 15 inches (38 cm.) for each eye. With a correction for reading of a +3 D. sphere for each eye, the near point of accommodation was 8 inches (20.4 cm.). Injections of diethylstilbestrol were stopped. Accommodation returned to normal in three months.

CASE 5.—Mrs. K. T., aged 57, had had her glasses changed four times by four ophthalmologists during the previous year. She was unable to see fine print at normal reading distance with her present glasses. No change in the lens or in the vitreous could be detected. There was no history or clinical indication of diabetes.

She had been taking diethylstilbestrol hypodermically and by mouth for the past four years, for relief of "hot flashes." An increased correction of 1 D. was required in her reading glasses. Medication was discontinued for two weeks, with complete recovery of normal accommodation for a person of her age.

In June 1942 the Council on Pharmacy and Chemistry of the American Medical Association [1] reported on diethylstilbestrol, citing several articles which noted undesirable reactions of the drug. The following symptoms, listed in the order of frequency of their occurrence, were stated to accompany the use of diethylstilbestrol: nausea, vomiting, headache, vertigo, abdominal distress, diarrhea and dermatoses. No mention was made of any ocular reactions.

Whether similar changes in the accommodation apparatus have occurred with the newer synthetic estrogens, such as triphenylethylene,[2] *aa*-di(paraethoxyphenyl)-*β*-phenylbromoethylene [3] and octofollin (2,4-di[parahydroxyphenyl]-3-ethylhexane),[4] I have been unable to determine. Just what physiologic changes occur in the accommodation apparatus with the use of diethylstilbestrol has not been worked out. It is felt that the report of these cases may induce others to study this phenomenon.

634 North Grand Avenue.

1. Diethylstilbestrol, report of the Council on Pharmacy and Chemistry, J. A. M. A. **119**:632 (June 20) 1942.

2. Robson, J. M., and Schönberg, A.: Estrous Reactions, Including Mating, Produced by Triphenyl Ethylene, Nature, London **140**:196 (July 31) 1937.

3. Robson, J. M., and Schönberg, A.: A new Synthetic Estrogen with Prolonged Action When Given Orally, Nature, London **150**:22 (July 4) 1942.

4. Blanchard, E. W.: Responses of Laboratory Animals to a New Synthetic Estrogen, Endocrinology **30**:1026 (June) 1942.

Obituaries

CARL FISHER, M.D.
1879-1944

Dr. Carl Fisher, one of the outstanding ophthalmologists of the West, died in Los Angeles on June 7, 1944, after a prolonged chronic illness.

I first met Carl Fisher when he came to practice his specialty in Los Angeles, in 1921. I well remember the great sense of pride we of the medical profession felt in welcoming to Southern California such a nationally known ophthalmologist, former chief of the eye, ear, nose and throat section of the Mayo Clinic (1909 to 1917). He brought to our community the best in ophthalmologic experience and professional tradition. He had recently returned from study in Europe, subsequent to active overseas duty in the Army in World War I.

CARL FISHER, M.D.
1879-1944

Carl Fisher was born July 16, 1879, in Breckenridge, Minn. He graduated in 1901 from Carlton College, Northfield, Minn., and received his degree of Doctor of Medicine from Harvard Medical School in 1905. His internships were at the Carney, the Massachusetts General and the Infants' Hospital, Boston; and his basic ophthalmologic training was at the Massachusetts Eye and Ear Infirmary. He left Boston in 1909 to accept the position at the Mayo Clinic, in which he became nationally known.

The medical societies of which he was a member included the American Board of Ophthalmology, the American Ophthalmological Society, the American College of Surgeons and the American Academy of Ophthalmology and Otolaryngology.

When he came to Los Angeles, these accomplishments led to his becoming an influential member of the local societies. He became clinical

professor of ophthalmology at the University of Southern California School of Medicine, a position he filled with active interest up to the time of his death.

Although not a prolific writer, his mental endeavors in the form of lectures, letters, discussions and clinical appraisals were gems of clear thinking, concise and pointed. Carl Fisher was blessed with one of the keenest intellects in the profession.

This attribute, plus his personal qualities, made him the ideal consultant, always contributing something of value to a solution of the problem and having that unusual knack of self effacement which allowed him to help the physician more than to impress the patient.

Because of his wisdom, common sense and unprejudiced desire to understand conflicting points of view, he was the final arbitrator in disputes and dissension arising within the local societies. He had a mild manner, inconsistent with that of an ardent crusader, preferring to exert his influence through subtle suggestion and exemplary professional conduct. It was characteristic of him to be tolerant of points of view relative to practice which, as judged by his own conduct, were distasteful to him.

He was thoroughly human, enjoying life fully and possessing a delightful sense of humor. This made him the preferred toastmaster and impromptu speaker. His repertory seemed inexhaustible and invariably reflected his sensitive and cultured tastes. He was a devotee of activities in art and music in the community.

One of his chief local interests was the Research Study Club of Los Angeles, of which he was one of the founders. He always attended the formal meetings and took an active part in the informal gatherings that followed. At these there was much jocular repartee, in which he had no peer. It was on such occasions that his culture, high ideals and way of enjoying life were manifested to his colleagues and made their mark on the younger men of the organization.

Carl Fisher's philosophy of life can best be portrayed through a chance remark made to me while visiting him at the hospital the day before his death. We were talking about the war, the coming invasion and places in France he knew so well, when he said something to this effect: "You know, it is strange that I, who am about to become an estate so soon, should be so interested in all the trivialities that occur about me. I really can't help being as curious and as interested as I ever was."

To those of us who knew him he will never be "just an estate," but one whose friendship enriched our lives by teaching us how to live. Perhaps the fact that he never married enabled him to give more freely of his friendship and time to the welfare of his colleagues than would otherwise have been possible.

A. RAY IRVINE.

News and Notes

PERSONAL NEWS

Appointment to University of Oregon Medical School.—Dr. Kenneth L. Swan, who for the past five years has been connected with the medical department of the State University of Iowa College of Medicine, has been made associate professor of ophthalmology at the University of Oregon Medical School, and assumed his new duties on July 10.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Congenital Anomalies

A CASE SHOWING PARTIAL DEFICIENT FUSION OF A MAXILLARY PROCESS WITH LATERAL NASAL PROCESS ON ONE SIDE. G. E. DODDS, Brit. J. Ophth. **27:** 414 (Sept.) 1943.

A Negro girl aged 3 months presented the congenital abnormalities to be described. The outstanding defect was distortion of the right nostril with a hiatus showing the interior of the nose, the cleft extending upward to within 1 cm. of the inner canthus, which had a wide, obtuse angle. There were small notches, or colobomas, in both upper lids and a notch in the right lower lid. There was no caruncle, and the presence of a plica semilunaris was doubtful. On the inner side of the medial canthus of the left eye there was a vertical hole, in appearance like the opening of a sinus. The cleft, or fissure, extended from the lower and lateral margin of the right nasal bone to a point where normally the nostril joins the lip. This was merely a cleft in the cartilaginous part of the nose. The eyes were normal in every respect.

A sketch accompanies the article.

W. ZENTMAYER.

Cornea and Sclera

HEREDITARY CORNEAL DYSTROPHY. J. R. MUTCH, Brit. J. Ophth. **28:** 49 (Feb.) 1944.

The subject of hereditary corneal dystrophy is considered fairly exhaustively. It is pointed out that Groenouw first described the disease in 1890 under the term "nodular corneae." That the disease is familial was first noted by Fuchs, in 1901. Mutch advocates the use of the descriptive term "granular" dystrophy and suggests that the term "nodular" be abandoned because it has caused so much confusion. He gives a pedigree in which 24 members were affected; of the affected, 14 were males and 10 females; the youngest was a boy aged 12 and the oldest 90 at the time of examination. Eight of the affected members were over 60 when examined. Fourteen of the patients were myopes. Mutch is inclined to think that the low myopia or myopic astigmatism was due in some cases to the corneal lesions but that in others the myopia was due also to heredity and was connected with the corneal disease. Up to the age of 50 corrected vision was in no instance less than 6/12. The visual acuity, however, does not suffer an irresistible diminution with the advance of age, as 4 affected patients, well over 60, still have vision of 6/12 or better. Only 1 patient, a man over 90, could have been certified as blind. In an old granite polisher the corneas had the appearance of hereditary dystrophy. The lesions, however, were all on the surface, but appearance of the eye and the distribution of the opacities were exceedingly similar. The lesion is not granular corneal dystrophy if it is unilateral, shows signs of vascularity, opacities are visible on examination with the naked eye in a patient under 30 and there is absence of a similar condition in both parents. The author considers the condition as probably a nutritional disturbance. The cornea receives its nutrition from the vascular loops at the limbus, lymph permeating centrally between the layers of the corneal lamellas. Any error in this circulation of lymph would affect the area farthest from the supply, namely, the central region of the cornea. Weight is given to this argument by the observation of several investigators (Bucklers), who stated that considerable improvement followed an intercurrent inflammation with vascularization of the cornea.

The progress of the disease was not brought to a standstill with certainty with any form of treatment used; far less was the disease made to retrogress. In certain cases the attempted cure was more harmful than the disease.

The refractive error should be corrected with glasses and the refraction periodically checked. The general health should be kept at as high a level as possible, with careful attention to diet, supplemented by the periodic addition of vitamins A and B.

The article is illustrated, and an excellent bibliography is appended. W. ZENTMAYER.

General

AN OPHTHALMOLOGIC REVIEW OF MORE THAN TWENTY THOUSAND MEN AT THE ALTOONA INDUCTION CENTER. L. P. GLOVER and W. R. BREWER, Am. J. Ophth. **27:** 346 (April) 1944.

Glover and Brewer present a table of refractive and congenital defects, traumatic injuries and ocular diseases. They give the following summary:

"Of 21,446 men going through the Altoona Induction Station, a total of 21.36 per cent had eye defects that lowered vision to 20/40 or less. This, excepting hyperopia, represents a fair cross section of eye defects in central Pennsylvania."

W. S. REESE.

General Diseases

OCULAR ROSACEA AND ARIBOFLAVINOSIS. W. M. FISH, Am. J. Ophth. **27:** 354 (April) 1944.

Fish reviews and compares his work with that of Wise and of Johnson and Eckardt, in general agreeing with Wise that rosacea is not a manifestation of riboflavin deficiency.

W. S. REESE.

Injuries

WAR INJURIES OF THE EYE: TRAUMATIC PROLIFERATIVE CHOROIDITIS DUE TO DOUBLE PENETRATING FOREIGN BODY. I. C. MICHAELSON and J. KRAUS, Brit. J. Ophth. **27:** 449 (Oct.) 1943.

In 6 of 7 cases of injury to the eye by a foreign body with an exit wound in the posterior part of the globe, the fundus had the following appearance after the vitreous had cleared sufficiently to permit detailed ophthalmoscopic study: A white mass of varying size could be seen in the area of choroidoretinal atrophic change. The whiteness was often milky and was sometimes glistening, with a few dots of crystalline brightness on the surface. The mass looked solid and in some cases projected far into the vitreous. It was not usually crossed by retinal arterioles or venules. Dark pigment might cover a part of its surface.

In the seventh case the fundus could not be seen on ophthalmoscopic inspection. In 2 cases in which pathologic studies were made the clinical appearance at the posterior wound appeared to be based on a massive proliferation.

The article is illustrated.

W. ZENTMAYER.

FIRST AID TREATMENT OF INDUSTRIAL EYE INJURIES. R. M. DICKSON, Brit. J. Ophth. **27:** 544 (Dec.) 1943.

The great majority of the injuries were caused by a fragment of steel or iron in the cornea, but brass, copper, aluminum and gun metal also played their part. Next in importance were foreign bodies from abrasive wheels. Burns from acids and sodium hydroxide were common in chemical factories, and there were a few cases of conjunctivitis from mercury fulminate. The author gives the following summary:

"This report deals with 2,478 eye injuries in 30 collieries in Scotland extending over a period of eighteen months, and 11,953 eye injuries in 30 factories in Scotland in six months.

"First aid treatment was essentially the use of 10 per cent albucid soluble (sodium sulphacetimide) as eye drops.

"Of the total injuries in collieries, 96 per cent returned to work with no loss of working time.

"In factories and shipyards, there was no loss of working time in 98.87 per cent of the cases.

"These eye drops have already been recommended by the Ministry of Fuel and Power for use as first aid treatment in collieries throughout the country.

"The excellent results after six months' trial in factories fully justify the recommendation that the new eye drops be adopted in all forms of industry where there is any danger of injury to the eyes."

W. ZENTMAYER.

Neurology

THE OCULAR MANIFESTATIONS OF SPONTANEOUS SUBARACHNOID HEMORRHAGE. A. J. BALLANTYNE, Brit. J. Ophth. **27:** 383 (Sept.) 1943.

Since in the majority of cases of subarachnoid hemorrhage the hemorrhage comes from the vessels of the circle of Willis, being due to the rupture either of an aneurysm or of a weakened portion of the vessel wall, the effusion of blood may be relatively slight but is likely to recur. The chief symptom in such cases is severe, deep-seated pain, often accompanied by paresis of one or another of the ocular muscles. Arteriographic studies of the brain may reveal the presence of an aneurysm, and lumbar puncture may disclose blood in the cerebrospinal fluid.

In the cases of severe hemorrhage, such as those reported in this paper, the clinical picture is entirely different. The patient is seen in a more or less comatose state, with a history of sudden onset and with signs suggestive of cerebral compression and meningeal irritation; pupillary anomalies, nystagmus, conjugate deviation of the eyes and paralysis of cranial nerves, including those supplying the extraocular muscles are often observed. These signs are not in themselves sufficient to justify a diagnosis of subarachnoid hemorrhage. The presence of retinal hemorrhages and papilledema, if observed on ophthalmoscopic examination, strongly supports such a diagnosis and a history of minor attacks adds to its probability, but the matter is settled by the discovery of blood in the cerebrospinal fluid.

Five fatal cases of spontaneous subarachnoid hemorrhage are described with special reference to the pathologic condition of the eye and its related structures.

The discovery of hemorrhage in the subarachnoid space at the base of the brain, within the sheaths of the optic nerve, and in many cases in the retina and even in the vitreous, originally led to the belief that the blood passed in a continuous manner in the subarachnoid space through the optic canal into the orbit and thence either along the course of the central retinal vessels or by lymph channels accompanying the optic nerve at the periphery of the lamina cribrosa into the retina.

It is not generally agreed that the blood in the cerebral subarachnoid space does not, as a rule, travel through the optic foramen into the subarachnoid space of the optic nerve. It is

more generally assumed that hemorrhage occurs in the dural sheath at the apex of the orbit and tracks forward into the subdural and the subarachnoid space, and that blood in the subarachnoid space, by exerting pressure and tension on the central retinal vein where it crosses the space, causes stasis in that vein and thus accounts for the hemorrhages in the retina and vitreous.

On the basis of the 5 cases described here, it is shown that hemorrhages occur over a much wider field than the optic nerve sheaths, the retina and the vitreous and that these hemorrhages cannot be accounted for by the assumed pressure on the central retinal vein.

The hemorrhages varied in distribution from case to case but were noted in and between the sheaths of the optic nerve, in the orbital fat and among the orbital muscles and around the posterior ciliary nerves, the ciliary ganglion, the ophthalmic and posterior ciliary arteries and the vessels of the chiasm and optic tracts, as well as beneath the retina, in all its layers, in front of the retina and in the vitreous. Serial sections showed that these hemorrhages were not continuous one with another but were discrete and independent, of simultaneous occurrence. It is submitted that the occurrence of such multiple hemorrhages can only be explained by a sudden rise of intracranial pressure, causing stasis in all the venous channels which drain the tissues of the eye and the contents of the orbit.

It is suggested that some of the clinical signs of subarachnoid hemorrhage, such as oculomotor pareses and disturbances of the conjugate movements of the eyes, may be explained by the occurrence in the midbrain of hemorrhages similar to those observed in the chiasm and the optic tracts in 1 of the cases.

W. ZENTMAYER.

ATAXIC NYSTAGMUS: A PATHOGNOMONIC SIGN IN DISSEMINATED SCLEROSIS. W. HARRIS, Brit. J. Ophth. **28:** 40 (Jan.) 1944.

Harris gives the following description of the sign: "When the eyes are turned laterally, the conjugate action appears weak, so that the inner eye, e. g., the right eye when looking towards the left, does not reach the inner canthus, but the outer eye shows coarse nystagmus, the eye reaching the outer canthus with a quick movement, and a slower return, as though the outward movement could not be maintained, owing to weakness. Usually the nystagmus of the outer eye continues as long as the patient looks in that direction, the inner eye meanwhile remaining stationery, not having quite reached the inner canthus. It is this discrepancy between the movements of the two eyes which suggests to the author the term 'ataxic nystagmus.'

"Usually this peculiar nystagmus will be present when looking either to the right or left, though it may be more marked in one lateral movement than in the other, or it may be seen only in one lateral direction."

W. ZENTMAYER.

Orbit, Eyeball and Accessory Sinuses

A CASE OF MONOCULAR HYDROPHTHALMIA, WITH SPECIAL REFERENCE TO ITS POSSIBLE RELATION TO THE STURGE-WEBER SYNDROME. A. GARROW and A. LOEWENSTEIN, Brit. J. Ophth. **27:** 335 (Aug.) 1943.

A case of monocular hydrophthalmia in a girl aged 11 years is described. She was first seen at the age of 5½ years, when the condition was typical hydrophthalmia, involving the right eye. Cyclodialysis and two trephinations were done at various times, with resulting hypotonus, but later the eye was removed because it began to shrink. Besides the many changes due to the hydrophthalmia, three angiomas were noted—the first in the choroid at the posterior pole surrounding the optic nerve, the second in the retina near the optic nerve and the third in the anterior part of the eye in connection with the choroid.

The authors discuss the relation of such a condition to the Sturge-Weber syndrome and suggest that angioma of the choroid may be the connecting link between the two diseases.

Calcification of the ocular tissue (capillaries, angiomas, internal limiting membrane and ganglion cells) was a feature of the case.

Reference is made to the allied anomalies—von Recklinghausen disease, Treacher Collins–von Hippel–Lindau disease and Bourneville's disease.

An angioma of the retina recently observed in a case of Bourneville's disease is briefly described.

The article is illustrated with photomicrographs of the pathologic features.

W. ZENTMAYER.

Physiology

THE SIGNIFICANCE OF THE DISTRIBUTION RATIOS OF NON-ELECTROLYTES BETWEEN PLASMA AND THE INTRA-OCULAR FLUID. STEWART DUKE-ELDER and H. DAVSON, Brit. J. Ophth. **27:** 431 (Oct.) 1943.

The authors state that for readers to whom a mathematical argument has no appeal the general problem with which this article deals may be stated thus: If the concentration of a given substance, urea for instance, is higher in the blood than in the aqueous humor, the system, plasma–aqueous humor, is not in equilibrium, and hence diffusion of urea should take place into the aqueous humor until the condition of equilibrium of equal concentrations in the two fluids is achieved. Kinsey and Grant admitted that this would happen in the absence of drainage and secretory activity. However, they

claimed that as soon as drainage occurs the concentrations become different; i. e., the system is no longer in equilibrium so far as urea is concerned, as defined thermodynamically.

The second law of thermodynamics states that a system of this kind can be maintained at a position of nonequilibrium only by the continued performance of work on it. Drainage of fluid away from the anterior chamber is not capable of performing the necessary work on the system, so that in the absence of secretory activity the differences in concentration claimed by Kinsey and Grant are excluded. W. ZENTMAYER.

Retina and Optic Nerve

THE HEREDITARY MACULAR DEGENERATION. R. I. LLOYD, Am. J. Ophth. **26:** 499 (May) 1943.

Lloyd describes four types of hereditary macular degeneration. In the first, and most common, form large white spots are scattered about the macula, and the condition is described in the literature as honeycomb, or guttate, choroiditis. The second type, in which many small white dots are scattered about the fundus, most numerous in or limited to the periphery, is usually described as retinitis punctata albescens. The third type appears under the term juvenile maculocerebral degeneration. A fine pigment is seen in the macular area, with thinning of the choroid. In the fourth type the appearance is that of disappearance of some of the layers of the choroid. The edges of the defect are sharp; the sclera is not bared, but long, straight choroidal vessels are uncovered. In some cases there is little pigment, and in others clumps of heavy pigment are present. The peripheral part of the choroid may resemble gyrate atrophy of the choroid and retina, or a type of choroideremia.

The article is illustrated. W. ZENTMAYER.

TWO CASES OF VISIBLE EMBOLI IN RETINAL ARTERIES, WITH A HISTOLOGICAL STUDY IN ONE CASE. W. E. C. DICKSON, G. C. PRITCHARD, L. H. SAVIN and A. SORSBY, Brit. J. Ophth. **28:** 1 (Jan.) 1944.

After a review of the clinical histories of the 2 cases in which actual emboli could be seen in the retinal arteries, the authors discuss several points in the cases. They believe that the white background was due to ischemic necrosis of the ganglion cell and nerve fiber layers rather than to mere edema and that massage shifted the embolus in the first case. In the second case the patient claimed subjective improvement in vision, but no accurate estimate was possible. Histologic examination of the eye of the second patient showed round cell infiltration of the optic nerve, choroid and retina, of the subacute or chronic variety; the block in the artery was an embolus, of some duration, lodged in a vessel with a slight or moderate degree of previous thickening of its tunica media and adventitia, but not of its intimal coat. There were localized proliferative thickenings of the intima in the immediate neighborhood of the embolus, which was just beginning to be canalized by the invasion of endothelial buds in its periphery. The changes in the retina, both proliferative and irritative, were not extensive and were most evident in the inner layers. These changes probably dated from the time of impaction of the embolus. There were recent acute changes in the choroid. The histologic evidence by itself was conclusive enough, but taken together with the clinical and pathologic evidence as a whole—the cardiac lesion with vegetations, the multiple embolic processes and the absence of arteriosclerotic changes and thrombus formation—leaves no valid doubt as to the nature of the block.

The article is illustrated. W. ZENTMAYER.

Tumors

CANCER OF THE EYELID. L. HOLLANDER and F. J. KRUGH, Am. J. Ophth. **27:** 244 (March) 1944.

This article represents a study of 125 cases of cancer of the lid. The impression was gained that most satisfactory results were obtained from scalpel excisions or contact roentgen irradiation, with use of a special shield.

W. S. REESE.

TUMOR OF THE LACRIMAL GLAND. J. J. FLICK, Am. J. Ophth. **27:** 362 (April) 1944.

Flick reports a case of tumor of the lacrimal gland which was incompletely removed by means of an osteoplastic flap over the left frontal lobe. The pathologist reported that the neoplasm showed features of adenocarcinoma.

W. S. REESE.

OPTIC-NERVE ATROPHY IN MALIGNANT NASOPHARYNGEAL TUMORS. M. R. FOLK Am. J. Ophth. **27:** 373 (April) 1944.

Folk reaches the following conclusions:

"Involvement of the optic nerve in malignant tumors of the epipharynx is not frequent.

"The optic atrophy in the cases herein described is preceded by invasion of the epipharyngeal tumor into the sphenoid sinus. This fact explains why the appearance of the optic atrophy is late.

"From the sphenoid sinus the malignant tumor may invade the optic nerve after perforating the lateral wall of the sphenoid sinus.

"The dural sheath of the optic nerve as well as the periosteum of the orbit and the dura is more resistant to aggression by the tumor than is bone tissue. This holds true for sarcoma."

W. S. REESE.

Visual Tracts and Fields

THE FORM AND CHARACTER OF ROD SCOTOMETRY. P. C. LIVINGSTON, Am. J. Ophth. **27:** 349 (April) 1944.

Livingston gives the following summary:

"Scotometry, employing self-luminous test objects and a red fixation light, appears to reveal defects in the central field of vision which cannot readily be found by customary procedures. It is well to confine this method to the 30-degree screen. Up to this point, it is possible to work with very low luminosities, such as 4×10^{-9} or 6×10^{-9} candle power, without introducing physiologic effects likely to be confused with early pathologic conditions. Further work is being undertaken to improve equipment and technique, so as to establish normal field characteristics."

W. S. REESE.

Vitreous

DISCISSION OF TRAUMATIC HYALOID DIAPHRAGM. J. FOSTER, Brit. J. Ophth. **27:** 462 (Oct.) 1943.

A bomb explosion caused multiple perforating injuries to both eyes due to fragments of glass, resulting in loss of the left eyeball and reduction of vision in the right eye to good light projection. In the right eye the lens was clear. Examination of the fundus showed a large hemorrhage and detachment of the retina in the nasal area. Later a gray haze behind the lens prevented a further view of the fundus. A membranous diaphragm being suspected, several months later the membrane was divided from in front with a Ziegler knife. Cataract subsequently developed. Two discissions were made, and ultimately vision with correction was 6/5. Almost two years later the condition of the eye was unaltered.

W. ZENTMAYER.

Therapeutics

THE SULPHONAMIDES IN EXPERIMENTAL OCULAR INFECTIONS. M. KLEIN and A. SORSBY, Brit. J. Ophth. **27:** 241 (June) 1943.

Klein and Sorsby give the following summary:

"The problems involved in the experimental study of ocular chemotherapy are indicated, stress being laid on the absence of a satisfactory technique for the organisms commonly met with in ocular infections and on the levels of concentration that different sulphonamides reach and maintain in ocular tissues by the general and local administration of these drugs.

"A review of the literature on experimental chemotherapy by general and local administration of the sulphonamides is given.

"Work is recorded which substantiates the claims for sulphanilamide in controlling experimental intra-ocular infection with Streptococcus haemolyticus and possibly also pneumococcus. B. pyocyaneus infections of the cornea were not controlled either by the general administration of sodium sulphacetamide or by the use of this drug in a variety of ways as a local application.

"In the absence of a more standard technique of inducing experimental infection of the cornea, the problem of the relative value of general and local use of the sulphonamides remains unsolved. The value of local therapy is still an open question."

W. ZENTMAYER.

Society Transactions

EDITED BY DR. W. L. BENEDICT

AMERICAN OPHTHALMOLOGICAL SOCIETY

JOHN GREEN, M.D., *President*

WALTER S. ATKINSON, M.D., *Secretary*

Seventy-Ninth Annual Meeting, Hot Springs, Va., June 10-12, 1943

Further Experiences with a System of Intracapsular Extraction of Cataract. DR. DANIEL B. KIRBY, New York.

This article was published in full, with discussion, in the April 1944 issue of the ARCHIVES, page 302.

Keratoconjunctivitis Sicca. DR. SANFORD R. GIFFORD †, DR. IRVING PUNTENNEY (by invitation) and DR. JOHN BELLOWS (by invitation), Chicago.

This paper was published in full in the August 1943 issue of the ARCHIVES, page 207.

DISCUSSION

DR. WILLIAM P. BEETHAM, Boston: Up to 1934 only 40 cases of filamentary keratitis had been reported. This paper shows how easy it is to see something when one begins to look for it. In my opinion filamentary keratitis is just another form of keratoconjunctivitis sicca. In 1934 I described a method of destroying the lacrimal puncta in an attempt to make better use of what tears are available. I found it difficult to keep the canaliculi closed. I used the actual cautery and the diathermy current, and I found that after weeks or months the canaliculi became recanalized; so during the past few years I have resorted to Dr. Verhoeff's suggestion of excising the lateral half of each canaliculus.

In applying the Schirmer test for tearing, I have always inserted the filter paper over the lacrimal puncta, and I have set as a standard 15 mm. of moistening in five minutes. I always measure the 5 mm. which I tuck into the conjunctival sac.

Dr. Gifford did not mention his criteria for destruction of the canaliculi. It has been my impression that if there is moistening of 10 mm. or more in five minutes the patient will be comfortable without resort to surgical treatment, but if the moistening is less than 10 mm., I suggest destruction of the canaliculi by excision of the external half of each.

† Dr. Gifford died February 25.

It is interesting to me that this condition is seen chiefly in females. In all of the 40 cases I found reported in the literature up to 1934 the patient was a female. In the papers by Sjögren and Bruce and in Dr. Gifford's paper, there have been a few cases of males. In all the cases I originally reported the patients were females, and I know of no case of the disease in a male, but I think I have been missing some cases of the mild form which Dr. Gifford has described.

DR. RALPH I. LLOYD, Brooklyn: I wish to speak of 2 cases of this condition. Case 1 was that of a young girl on whom Dr. Charles Rosenthal operated for dermoid just beyond the lateral rectus muscle. The mass extended well down into the orbit, and healing was uneventful. A month later the characteristic symptoms of filamentary keratitis appeared. These were promptly relieved by use of the moist chamber spectacle, which she lays aside now and then, only to have prompt return of all the annoying symptoms. This case suggests that damage to the ciliary ganglion caused the disease.

The moist chamber spectacle will do all that surgical closure of the canaliculi will do, and I expected that Dr. Beetham would mention the device, as it seems to have originated in Boston. Locke's solution, buffer solutions and castor oil have all failed to help the few patients I have treated.

Case 2 was that of a single woman aged 45 who had been troubled for about a year with what was diagnosed as keratitis sicca. The Schirmer test showed the same rate of secretion (7 mm.) in the apparently normal eye as in the offending eye. The moist chamber spectacle is of great comfort to her, but every now and then a relapse occurs, at which time fine filaments are seen on the cornea. The only associated conditions are the menopause and a cutaneous condition, which has not yet been diagnosed.

DR. ARNOLD KNAPP, New York: It was formerly taught that removal of the lacrimal gland was feasible and that the action of the mucous cells in the conjunctiva was sufficient to preserve the integrity of the cornea; in fact,

at one time the operation of removal of the accessory lacrimal gland was a popular one for relief of tearing after extirpation of the lacrimal sac.

Did Dr. Gifford observe a definite lesion of the conjunctival epithelium in his cases?

DR. ALBERT L. BROWN, Cincinnati: I should like to cite several cases of this interesting condition, on the basis of a possible etiologic factor. In 1 case the condition developed after an operation on the gasserian ganglion, the entire cornea becoming involved about five weeks later. In another case it followed direct trauma to the cornea from a scratch by a dog's nail. This accident caused a minor abrasion, apparently rather superficial, which cleared in a reasonable time, from ten days to two weeks; in about three weeks the cornea began to break down and presented a typical picture of dry cornea, with diminished lacrimal secretion. In 3 cases of mixed tumor of the lacrimal gland no sign of corneal involvement appeared after operation; in 1 case, in particular, I removed a great proportion of the gland five years ago, but the cornea has remained clear.

DR. LAWRENCE T. POST, St. Louis: I wish to speak of those cases in which there are corneal changes and the Schirmer test shows not more than 1 mm. of dampness on the exposed filter paper. I have been so much impressed by the presence of a glandular deficiency in most of these cases that I have made it a routine to prescribe a glandular preparation, theelin for the women and testosterone for the men, in the relatively small group of patients I have treated. In addition, I have given vitamin A routinely, 50,000 units a day, and have instilled haliver oil in the eye three times a day. A woman with a particularly severe form of the disease, who had had to stay in a dark room for three years and had not been able to carry on any work because of her eyes, was much relieved by the intravenous injection of antimony and potassium tartrate. She was given injections of this substance because of its known beneficial effect on epithelium. I have had her under observation since, for four or five years, during which time she has been able to work and has been relatively free from discomfort.

DR. FRANCIS H. ADLER, Philadelphia: Tears are equivalent to about a 1.4 per cent solution of sodium chloride. My associates and I have not had enough cases of this condition to try hypertonic solutions. In the cases we have had we have used an artificial solution of tears comparable in tonicity to blood; in view of Cogan's work, however, I suggest the use of a hypertonic solution, which is equivalent to a 1.4 or 1.5 per cent solution of sodium chloride.

DR. NELSON M. BLACK, Miami, Fla.: In the investigation of these cases, did Dr. Gifford make any attempt to determine the blood picture and the basal metabolic rate?

DR. SANFORD B. GIFFORD, Chicago: I am glad that Dr. Beetham was able to discuss the paper, for, as every one knows, he was the first to close the canaliculi in order to relieve keratitis sicca. I agree with him in almost everything he said except with respect to the method of making the Schirmer test. When I looked up Schirmer's description of the test, I found that he did it as I have indicated. Dr. Beetham records the 5 mm. of the paper that is turned over. That portion is almost always wet. I measure from the border of the lid down; if he were to set 10 mm. of wetting as his criterion for destruction of the canaliculi, it would correspond to the 5 mm. as I measure it; this is the criterion I use, except that I also take into consideration the clinical picture of my patient. The patients of group I showed a slight lacrimal deficiency, with no corneal changes, and those of group II, slight corneal changes; so treatment depends chiefly on the severity of the corneal lesions associated with a lacrimal deficiency of 5 mm. on repeated tests. As a therapeutic test the puncta may be closed temporarily with trichloroacetic acid. There is no increase in lacrimal secretion after destruction of the puncta, but retention of the tears in the sac gives the patient great relief.

A moist chamber spectacle may be of value in cases of extreme deficiency of tears. Our patients never get complete relief, but it is remarkable how much relief they do obtain. They are always a little sensitive to light. I have a patient now—with the worst condition I have ever encountered—who is getting only moderate relief. Her vision is reduced to 20/100 as a result of the repeated occurrence of small corneal lesions; these have not healed after closing of the puncta. Some patients have permanent scarring. While the lesions are epithelial, they seem to produce notable reduction in vision.

Dr. Knapp asks about the epithelium: I have not made any sections but can see the thickening. It resembles that in trachoma. The epithelium must show a pathologic change.

Dr. Post mentioned glandular therapy. In my paper I described the use of theelin and thyroid for a few patients who showed a low metabolic rate, and Dr. Puntenney made a complete chemical analysis of the blood in a few cases. He did not find anything of significance except an increased sedimentation rate, with moderate anemia in some cases. He also made examinations for the vitamin A content of the blood and found no deficiency. However, we give vitamin A by mouth, as Dr. Post does. We do not think it has any value in local treatment.

I am surprised that in Dr. Brown's cases of tumor of the lacrimal gland keratoconjunctivitis sicca did not follow operation; that can be explained only on the ground that some of the gland, either the accessory or the main gland, or at least some of the ducts, must have been left. One usually has complete keratitis sicca, as I did in a case of lymphosarcoma, in which it was necessary to remove some of the conjunctiva in that region.

Dr. Adler's suggestion is a good one, and I shall try it.

Subjective Experience with Epidemic Keratoconjunctivitis. Dr. Thomas D. Allen, Chicago.

I infected my own eyes in a moment of preoccupation. Vision deteriorated during a period of three weeks until it was 20/200 or less in each eye. The physical signs were classic and were confirmed by a number of the members of this society. Treatment was of no avail until a preparation of vitamin C was given intravenously.[1] Vision was slowly restored to normal. The psychologic reaction was at first disturbing.

DISCUSSION

Dr. F. C. Cordes, San Francisco: Epidemic keratoconjunctivitis appeared for the first time in San Francisco in August 1941. There is no question that the disease is contagious. In California it is known as "shipyard conjunctivitis" and is considered an industrial disease. In my opinion, it is so called because it occurred in persons working in close quarters in the shipyards, where one shift went off as the next came on and the men handled equipment which had previously been contaminated. The severity of the keratitis varies tremendously. In some patients it lasts a relatively short time and clears up without difficulty. With others it has taken a year and a half for the opacities to clear sufficiently for vision of 20/20. Of the patients I have seen, there is 1 whose vision has not improved beyond a questionable 20/40. I tried all types of therapy, without any apparent benefit. Dr. J. W. Crawford, who has seen the largest number of patients in San Francisco, had a similar experience and stated that the use of cold compresses gave the greatest relief. Patients with the more severe type of keratitis seem to retain changes visible with the slit lamp, even though vision has returned to normal. These changes consist of fine annular scars, similar to those described in cases of keratitis nummularis.

Dr. John N. Evans, Brooklyn: For two years I have been using a preparation of 1 per cent paredrine hydrobromide with 2 per cent boric acid for a variety of pathologic disturbances of the epithelium, such as bullous keratitis, superficial punctate keratitis and keratoconjunctivitis. It seems to me that I have gained a therapeutic advantage by so doing. I have not observed any opacities in a small number of cases of "shipyard conjunctivitis." This may be a coincidence. I am not making a positive statement as to the value of this treatment, but I suggest its trial. I have had 3 cases of so-called virus pneumonia in the immediate families of patients with epidemic conjunctivitis.

Dr. Peter C. Kronfeld, Chicago: Except that a colleague and friend was concerned, Dr. Allen's case was not unusual. It emphasizes the important fact that the onset of epidemic keratoconjunctivitis may be gradual and noncharacteristic. During the first two days of Dr. Allen's stay at the hospital my associates and I were not at all sure of the diagnosis. On the third day the situation had become obvious.

In this connection, it may be of interest to cite the histologic report by Günther (*Klin. Monatsbl. f. Augenh.* **103:** 309, 1939). A patient one of whose eyes had been blind for a number of years as the result of a retinal detachment presented himself at an ophthalmic clinic in Berlin with bilateral epidemic keratoconjunctivitis, with the request that the blind, painful eye be enucleated. Histologically, subepithelial infiltrations and areas of degeneration of Bowman's membrane were noted. Günther stated that the latter lesion was primary.

Dr. Harold Henry Joy, Syracuse, N. Y.: A rather mild epidemic occurred in Syracuse. I saw about 25 cases. What impressed me in my series was the scattered distribution of the cases. Few were associated with any particular industry, and in only 1 instance did I know of direct infection from one patient to another.

In 5 cases the condition was unilateral, and in at least 3 of these the patient was not one who would be careful about protecting the uninvolved eye.

Dr. John H. Dunnington, New York: I have a patient with this disease who has been under my care for the past six months, and his vision is still 20/200. There have been several others whose corneal lesions have been present for as long as a year. I am convinced that some of the therapeutic measures employed in Dr. Allen's case must have had a beneficial effect.

Dr. C. A. Clapp, Baltimore: I raise the question of the possibility that the virulence of this infection subsided as it swept over the country. I have seen a large number of cases in Baltimore, chiefly among shipyard workers. The virulence has been much less than that reported in the West. There have been only occasional rise in temperature and little swelling of the preauricular

glands. The complications of the cornea have been comparatively slight, and the spread of the infection in families has been negligible.

DR. JONAS S. FRIEDENWALD, Baltimore: I should like to add a word to the suggestion that there may be a connection between this disease and virus pneumonia. In one of the hospitals in Baltimore two of the house officers made a bronchoscopic examination of a patient with virus pneumonia, after which both had a conjunctivitis in the eye used with the bronchoscope which was not bacterial and which was thought to be related to keratoconjunctivitis.

DR. THOMAS D. ALLEN, Chicago: In answer to Dr. Evans' comment on the use of paredrine hydrobromide and the absence of corneal opacities in his cases, the incidence of corneal opacities varied from 15 per cent, in one epidemic, to about 80 per cent, in another.

Dr. Joy mentioned the unilaterality of the disease. I have seen it in one eye in many cases. I cannot see why it does not pass to the other eye in such patients, who are often dirty, unless there is an inherent tissue resistance.

Relation Between Bacterial Flora of the Conjunctiva and That of the Nasal Mucosa, with Special Reference to Certain Extraocular Inflammatory Diseases. DR. CONRAD BERENS and EDITH L. NILSON (by invitation), New York.

Eighty-three bacteriologic studies were made of the conjunctival and the corresponding nasal membranes of 75 patients. In vitro tests for toxicity, the results of which correlated with the results of tests on animals for pathogenicity, were used to determine individual strains of streptococci and staphylococci. In 50 per cent of the cultures there was a notable relation between the nasal and the conjunctival flora; in 43.3 per cent cultures of nasal secretions yielded "toxic" organisms, with negative results for cultures of secretions from the eye; in approximately 3 per cent cultures of material from the eye yielded organisms of greater toxicity in the eye than did cultures of nasal secretions, and in only 3 per cent were the bacterial flora of the conjunctiva and those of the corresponding nasal membranes entirely unrelated.

The bacteriologic relation between nasal and conjunctival infections is substantiated by unilateral clinical symptoms, and in many cases the nasal exacerbations coincide with, or are closely followed by, ocular symptoms. These results suggest the advisability of examination of the nasal membranes as a possible source of infection in determining the cause of chronic and recurrent extraocular inflammatory diseases.

DISCUSSION

DR. PAUL A. CHANDLER, Boston: I believe this report is significant. It is well known that in cases of multiple boils all over the body the offending agent is nearly always present in the nose. Many people who have recurrent boils and stys have been relieved by scrupulously keeping the hands away from the nose. Persons who treat boils and carbuncles feel that a frequent cause of such infections on the neck is the patient's rubbing the back of the neck with the hand. One would be wise to point out to patients who have recurrent stys and ocular infections the possibility that infections may be transferred from the nose to the eye.

DR. CONRAD BERENS, New York: The mode of transference of infection is conveyance by means of the hands or the handkerchief. Since these ocular infections so often occur simultaneously with infection of the nasal membranes, and since in unilateral ocular conditions the infection in the eye "lights up" with an exacerbation of the nasal infection on the same side, my associates and I are inclined to believe that infections may be transmitted by way of the nasolacrimal duct, and not merely by the hand or the handkerchief.

Written Examinations for the American Board of Ophthalmology. DR. S. JUDD BEACH, Portland, Maine.

The American Board of Ophthalmology has not given written examinations since 1940. It is now devising questions broad enough to reveal qualifications of the candidates, yet sufficiently rigid to obstruct concealment of their weaknesses. In this report are described the development of written tests in twenty-five years of the board's existence.

Distribution of Certain Oxidative Enzymes in the Ciliary Process. DR. JONAS S. FRIEDENWALD, DR. HEINZ HERMANN (by invitation) and DR. ROBERT MOSES (by invitation), Baltimore.

Metastasis of Malignant Tumors to the Eye: Report of Two Cases. DR. EDWARD C. ELLETT, Memphis, Tenn.

Two cases of metastatic carcinoma of the choroid are reported in which the primary lesion was in the breast. The first was a case of generalized metastasis from a malignant melanoma of the abdominal wall, causing paresis of an external rectus muscle, and the second, a case of rubeosis of the iris, accompanying a malignant melanoma of the choroid.

DISCUSSION

DR. ALBERT N. LEMOINE, Kansas City, Mo.: In 1936 I reported a case of bilateral metastatic carcinoma of the choroid in which one eye was treated by roentgen ray therapy. Useful vision was retained for the rest of the patient's life. Recently, I had a patient who had been at the Mayo Clinic a year ago, where an exploratory operation was performed on the lung for a tumor, which proved to be an inoperable carcinoma. She was sent home for roentgen irradiation, and she has received such therapy to the chest off and on up to the present. At the time of this report the roentgenograms fail to show any growth in the lungs. Apparently, the treatment arrested the progress of the tumor. However, in January her vision began to fail, and in February it was still worse. I first saw her a month ago, when vision without correction was 6/200 in the right eye and 20/200 in the left eye; with correction it was 20/200 in the right eye and 20/100 in the left eye. In the right eye there was a large yellowish mass, as described by Dr. Ellett. The mass extended around the nasal part of the disk, and below and over to the macula. The highest point of elevation was 9 D. In the left eye there was a similar symmetric lesion, which was elevated about 3 D. Roentgen irradiation was immediately started. At present (I saw her three days ago) vision was correctable to 20/100 in the right eye and 20/50 in the left eye. The lesion in the left eye was completely flattened, and in the right eye it was still elevated 4 or 5 D. The visual field in the right eye was not improved. The upper quadrants were entirely obliterated; apparently the arterial circulation supplying that portion of the retina was occluded. In the left eye the visual field was almost normal. I feel it is important in such cases to use radiotherapy, especially when there is bilateral involvement.

DR. C. A. CLAPP, Baltimore: In the July 1926 issue of the *American Journal of Ophthalmology* I reported 2 cases of so-called metastatic carcinoma of the choroid. At that time only 118 cases had been reported in the literature. I wish to show slides of 1 of the cases and to tell a bit of the later history.

The patient, aged 36, had a carcinoma of the breast removed in 1922, and her condition was considered cured. In April 1925 she consulted me because of loss of vision in the left eye. I found that she had a detachment of the retina, and, because of darkness on transillumination and the previous history, I made a diagnosis of carcinoma of the choroid. Incidentally, the roentgenograms showed involvement of the occipital bone, the spinal column and the upper ends of both femurs. Under these circumstances, I did not advise operation for the detachment or enucleation of the eye. In July she returned with acute glaucoma in the same eye and in great pain. I therefore advised enucleation. The operation was performed on July 11. The first slide shows the growth extending over the posterior third of the eye; the next is a photomicrograph of the carcinoma which was removed from the breast, and the next shows similar carcinomatous cells from the choroid. About a month later she returned with detachment of the choroid in the other eye, which was also dark on transillumination, an indication of metastatic carcinoma in this eye. Incidentally, I believe Dr. Ellett did not mention that in about one third of the reported cases the condition was bilateral.

A Problem of Split Macula: Study of the Visual Fields. DR. JOHN N. EVANS and DR. E. JEFFERSON BROWDER (by invitation), Brooklyn.

This article was published in full in the January 1944 issue of the ARCHIVES, page 431.

DISCUSSION

DR. FRANCIS HEED ADLER, Philadelphia: The authors called this tongue-shaped scotoma an angioscotoma; I am puzzled to know what vessel in the macula would give rise to an angioscotoma.

DR. ALEXANDER E. MACDONALD, Toronto, Canada: On account of delusions, hallucinations and emotional instability, it was necessary to remove the right cerebral cortex of a white girl aged 16 years. This was done on June 17, 1936, and on July 17 vision was 6/9 in the right eye and 6/36 in the left eye, the latter having diverged and been myopic since childhood. dDr. H. M. Macrae reported that telescopic observations on the fixing eye showed no unsteadiness during examination of the fields and that at least 1 degree to the blind side of the hemianoptic fields of vision was preserved in each eye.

The patient made a good recovery and, after minor operations for long-standing contractures, was able to do housework and errands. Her temperament also improved.

DR. JOHN N. EVANS, Brooklyn: We did not mean to say that the central scotoma was an angioscotoma, although I believe part of it was. In this particular case we referred to a small "submacular" tongue and the little tongues above. They were used as controls. We could have mapped some of them farther and shown the branches. When a central scotoma is developing, with slight disturbances, such as afterimages, a tongue forms along the vessels which come from the optic nerve on the side of fixation. In every instance there seems to be extra activity

on the part of the circulatory mechanism, apparently in an effort to build up, repair or reestablish the function of the central region. Our studies showed that in normal subjects the tongues of angioscotoma never run more than 7 or 8 degrees from the region of fixation.

Retinal Detachment and Trauma. Dr. Arnold Knapp, New York.

This article was published in full in the December 1943 issue of the Archives, page 770.

DISCUSSION

Dr. Mark J. Schoenberg, New York: The first difficult point to settle is concerned with the relation of retinal detachment and trauma. It is important to consider whether the detachment develops only after direct trauma to the eyeball or whether it may appear after an indirect trauma or a "strain."

In taking the history of my patients, I have made a special effort to get minute details about any accident or unusual happening that occurred, not only immediately but weeks before the appearance of the detachment, with the intention of establishing whether the factor which produced the detachment was the sole or the precipitating cause.

Of course, most ophthalmologists would consider a direct blow on the eyeball sufficient reason for the development of a detachment; but many hesitate to believe that a long trip in a wagon or an automobile, a bump on the head, a fall, a violent jar of the body, vibratory treatment of the scalp, prolonged reading, a miss with a golf club (the patient hitting the ground violently instead of the ball), lifting of a heavy weight, attacks of violent sneezing or coughing or straining in defecation may also be capable of bringing about such a detachment. I am glad that Dr. Knapp is inclined to believe that detachment of the retina may develop after an indirect trauma. I have collected a number of instances of such indirect trauma from the histories of my own patients and I feel that often such complaints should not be brushed aside as trifling or unimportant. Indirect trauma may at times be the precipitating cause of detachment in eyes which have been predisposed by a disease preceding the trauma, such as choroiditis, cystic degeneration of the retina, degeneration of the vitreous and intraocular foreign bodies. In 9 per cent of my own patients complications of this sort have occurred. In 11 per cent the trauma was direct to the eyeball. In 45 per cent there was indirect trauma—a "strain," such as prolonged reading, sneezing or coughing. About 35 per cent of the patients gave no history of direct or indirect trauma.

The second controversial point to be discussed is the latent period which intervenes at times between direct trauma and the appearance of signs and symptoms of the detachment. I believe that in some cases the signs, and even the symptoms, of retinal detachment appear weeks, and even months, after the occurrence of the trauma. Some patients may have had vague symptoms, such as flashes or spots before the eyes, but they overlooked mentioning them to the examiner unless they were carefully questioned.

Of a number of interesting points suggested by the statistical study of retinal detachment, it is worth mentioning that 22 per cent of my patients had a low degree of myopia, of less than —1 D. sph., and that 16 per cent had bilateral detachment—approximately the incidence of 14 per cent (11 per cent males and only 3 per cent females) which Dr. Knapp mentioned. Further, unilateral detachment prevailed in the right eye in the males and in the left eye in the females.

Dr. John H. Dunnington, New York: The role of indirect trauma in the production of detachment of the retina is difficult to ascertain. I agree with Dr. Knapp that this possibility cannot be ignored, but I am not in complete agreement with Dr. Schoenberg, whose list of agents producing indirect trauma includes practically everything one might do, from sneezing to reading. From the standpoint of compensation, it is extremely important to make a notation on the presence or absence of trauma at the time of the initial examination. This prevents embarrassment when several weeks later the patient thinks of some vague indirect trauma he experienced several months prior to the onset of the detachment. It is my opinion that indirect trauma is of comparatively little importance in the production of detachment of the retina. As Dr. Knapp has pointed out, the frequency with which both eyes are affected is a strong argument against the importance of indirect trauma.

Dr. Jonas S. Friedenwald, Baltimore: I should like to comment on a minor statistical aspect of Dr. Knapp's report. In discussing the frequency of detachment in respect to age, he pointed out that the age group in which detachments are most frequent is that of 40 to 60, an observation which corresponds with the experience of all commentators. The implication that detachment is less of a risk in persons over 60 is, I think, incorrect, for the proportion of persons in the community as a whole who are over 60 is steadily decreasing, and if one compares in each age group the incidence of detachment with the incidence of survival of the population as a whole, one finds that the risk of detachment steadily increases with age. The same holds with respect to the incidence of high myopia among

persons with retinal detachment. It is quite true that the most frequent record of detachment is made in cases of a medium degree of myopia, say, between 5 and 15 D. That does not imply, however, that the risk of detachment for a person with 20 to 30 D. of myopia is less than that for a person with 10 D., since the frequency of the high myopia is much less. I should like, also, to comment on the long interval between injury and the onset of symptoms. No doubt the onset is often within a two week period, but there are cases, I believe, in which a much longer interval exists and in which the relation between the trauma and the detachment is at least strongly indicated. In this connection, I may report the case of a boy of 15, with hypermetropia, who was hit directly in the eye by a peach falling from a tree. He was unconscious for a minute or two. He had no visual symptoms after this accident, except during the few minutes of his recovery, until nine months later, when detachment, with an extensive disinsertion of the retina, developed. No other injury occurred during the interval, to his knowledge. Such a severe trauma, I think, cannot be disregarded as a cause of the detachment, even though the interval may be much longer than one ordinarily expects.

DR. ARNOLD KNAPP, New York: In answer to the question raised by the discussers of this paper, I confess that the question of indirect trauma is a riddle and that undoubtedly the length of time before the condition develops may be long; in other words, the latent period is variable. I had hoped, however, that some one who has made a special study of ocular movements would comment on the involuntary protective movements of the eye: In what way do they take place, and under what conditions? Furthermore, can increased abdominal or thoracic pressure affect the intercerebral pressure, and in this way influence the circulation of the eye?

So-Called Primary Retinal Tuberculosis: Sarcoidosis of the Retina. DR. ALEXANDER E. MACDONALD, Toronto, Canada.

Two unusual pathologic conditions in the retina are contrasted. In the first case, isolated tubercles were observed in the detached retina of a boy aged 5 years, whose chest appeared normal on roentgenographic examination and whose reaction to the cutaneous tuberculin test was negative. In the second case, scattered nodules of sarcoidosis (Boeck's sarcoid) were noted in the retina of a woman aged 50. In both cases enucleation was done after the diagnosis of a malignant growth.

DISCUSSION

DR. E. V. L. BROWN, Chicago: Dr. MacDonald has shown in his slides something which may be of help in differentiation of sarcoidosis and tuberculosis of the retina. In the first specimen there was a clear outline of a vesicle, or delimiting tissue, over part of the node. He pointed out an apparent separation from a central line of cells, a closing off in what might be a vessel from within a vessel. The second specimen, stained by the Verhoeff method, presented the same appearance. In the third specimen the sphincter of the iris was bent like a hook. It will be recalled that all capillaries in the iris lie behind the sphincter and that any explosive process from the capillaries would displace the sphincter in this way. In tuberculosis, on the other hand, the nodes occur at the bifurcation of veins, on the surface of the vessel. If this distinction is correct, the lesion develops inside the vessel in the case of sarcoidosis, exploding outward into the tissue in all directions, and on the surface of the vessel in the case of tuberculosis.

DR. ALEXANDER E. MACDONALD, Toronto, Canada: With regard to Dr. Brown's suggestion about the blood vessels, the lesions of sarcoidosis were in nearly every instance close to, but not in, the vessel.

In the last lantern slide in Dr. Lloyd's case, was the lesion in the choroid or in the retina?

DR. RALPH I. LLOYD, Brooklyn: It was in the retina, along one of the retinal blood vessels.

DR. ALEXANDER E. MACDONALD, Toronto, Canada: Was there any caseation?

DR. RALPH I. LLOYD, Brooklyn: The case was one of tuberculosis, as described by Fleischer. There was no caseation in the case, but autopsy was not performed.

Ophthalmoscopic Classification of Hypertensive Diseases. DR. GRADY E. CLAY, and DR. J. MASON BAIRD (by invitation), Atlanta, Ga.

Ophthalmology has played a significant part in the study of the various hypertensive states and is most important as a diagnostic aid. The present classification is not new, but one which has been used for years and has been found to be simple, and usually of diagnostic value for use in the medical wards.

Angiospastic hypertension is the earliest form, characterized by spasms of the arterioles. It is most common with toxemia of pregnancy and next most frequent with unilateral renal disease. The degrees of angiospasm may be estimated as grades I to IV. Ophthalmoscopically, one may grade the degree of sclerosis, and therefore the stage of essential benign hypertension. We classify arteriolar sclerosis as grades I, II, III

and IV. In essential benign hypertension no changes are present except the varying degrees of sclerosis; the appearance of edema, hemorrhages and exudates, we feel, is evidence of renal damage, and we designate such a condition of the retina as secondary nephritic hypertensive neuroretinopathy. Malignant hypertension, we believe, is a disease entity, characterized by severe arteriolar spasms, extreme edema and pronounced hemorrhages and exudates.

The changes in the fundus seen in patients with nephritis, especially chronic glomerulonephritis with associated hypertension, are designated as primary nephritic hypertensive neuroretinopathy.

DISCUSSION

DR. P. J. LEINFELDER, Iowa City: There is no question that the terminology in the literature is confused. Graduate students at the State University of Iowa College of Medicine repeatedly come with the complaint that they cannot make head or tail out of the classification of hypertensive vascular disease. As a result, my colleagues and I have had to form our own classification, which I am happy to say is in considerable agreement with Dr. Clay's. All may take exception to parts of his outline; yet for practical purposes it clearly expresses the state encountered in the study of large numbers of hypertensive patients.

I should like to speak of the exceedingly small arterioles, the branches of the central artery of the third and fourth order. Frequently the first changes are seen in these vessels and consist of local irregularities and narrowings, which may progress to such an extent that the small vessels are seen with extreme difficulty. In the earliest stages the changes of hypertensive vascular disease seem to be confined exclusively to these small vessels. As the disease advances, the vascular changes progress down the vessels toward the nerve head, and in the late stages they involve the largest arterioles.

DR. THEODORE L. TERRY, Boston: Section of certain sympathetic nerves and removal of functioning adenomas of the adrenal glands tend not only to control, but in some instances to cure, younger patients with severe hypertension. Surgeons, cardiologists and internists most active in selecting the hypertensive patients who should have surgical treatment know of the importance of careful, detailed ophthalmic examination, and they are as concerned as the ophthalmologists over the selection and use of proper terminology. They, too, should be consulted before a terminology is adopted, even though they already are guided to a great extent by the report of the ophthalmologist on the state of the fundus.

DR. JONAS S. FRIEDENWALD, Baltimore: Dr. Clay has brought up an important problem in emphasizing the difficulties in nomenclature in this field. It may be of help in the solution of this problem if one understands more fully how this confusion has developed. Two aspects seem to me to have contributed seriously to the development of confusion in the study, knowledge and classification of vascular diseases. First, vascular diseases have been classified primarily from two points of view: the clinical and the pathologic, and these two systems of classification have never been satisfactorily correlated. The pathologist recognizes at least two primary vascular diseases: the first, a disease of the intima, which consists of proliferation of the endothelium with fatty plaques, a change which was first called arteriosclerosis and later atherosclerosis, with other new names from time to time, and the second, the hyaline degeneration of the media of the artery. Since the second type occurs most often in the smaller vessels, it was originally called arteriolar sclerosis, and further names have been given it from that point of view. Both these conditions may, on occasion, affect vessels of the same size, so that classification of the two diseases on the basis of the size of the vessel attacked and of the retinal disease on the fact that the arterial vessels seen there are all arterioles does not in itself resolve the confusion. In some cases the major vessels in the retina may at some time show atheromatous changes and be associated with what used to be called atherosclerosis in the larger vessels of the body. In other cases they may show hyaline degeneration and be associated with arteriolar sclerosis or hyaline degeneration of the media of the vessels elsewhere in the body. Thus, the overlapping of these two diseases in the medium-sized vessels constitutes one of the barriers to a reasonable classification.

The second aspect of the difficulty in classification is that while either of these disease processes may be primary, each may indirectly cause the other to develop. For instance, if a condition begins with what used to be called arteriolar sclerosis, that is, hyaline degeneration of the media of the arterioles, hyaline degeneration of an arteriole in the vasa vasorum of a coronary artery may follow. The coronary artery suffers in nutrition as a result, and an atheromatous plaque may develop on the intima of that vessel and the patient die of a myocardial infarction. Thus, atherosclerosis may in many instances be secondary to the hyaline change in the small arterioles. Conversely, the condition may start with what is primary atherosclerotic disease; an atheromatous plaque may form in the renal artery, and ischemia of the kidney and secondary malignant hypertension and arteriolar changes in the small vessels may develop. That these two conditions are so confused in their course,

and that many patients in the terminal stage have both diseases, one primary and the other secondary, the one mutually fortifying the other, make the nomenclature confusing.

Dr. John N. Evans, Brooklyn: It seems to me that the problem goes back much farther than classification. One must begin with a better understanding of the basis on which inferences are drawn. One cannot directly justify a diagnosis of "vasospasm" from anything that is seen. What one can say is, "These vessels look narrow." Then one asks, "What are the many factors which can make vessels look narrow?" One may ask the medical student, "What are the distinguishing features of choked disk and papillitis?" It is easy to tabulate over one hundred factors and conditions which can produce blurring of the nerve head. If one cannot decide what features differentiate one of these conditions from the others, how is one going to make progress in classification? If the authors' classification is to be accepted, it will have to begin with acceptable criteria from which inferences may be drawn.

Dr. Grady E. Clay, Atlanta, Ga.: We consider benign hypertension by far the most common hypertensive disease. The chances are that if in the 10 cases presented here we had examined the fundi shortly before death, we should have seen only arteriolar sclerosis, probably of grade II. The large majority of patients with hypertension show a gradual increase in the degree of arteriolar sclerosis, and that degree of sclerosis represents a grade of hypertension, in our opinion. We believe that irregularities in the caliber of the arterioles indicate that there has been a previous spasm and that the patient has had a fluctuating type of hypertension.

I did not speak of the senile type. Senile arteriosclerosis does not fit into the group of hypertensive diseases. In the patient with senile arteriosclerosis sclerosis of the arteries usually exists prior to the development of arteriolar sclerosis. The arterioles are usually straighter than those of patients of the hypertensive group; the reflex stripe is not so conspicuous, and there is not the irregularity in the caliber of the arterioles. It is our observation that chorioretinitis is especially common in cases of senile arteriosclerosis. One sees persons between the ages of 70 and 80 who have normal arterioles. The occurrence of isolated hemorrhages in cases of senile arteriosclerosis is of no prognostic value.

Traumatic Enophthalmos. Dr. Raymond L. Pfeiffer, New York.

This article was published in full in the December 1943 issue of the Archives, page 718.

DISCUSSION

Dr. John Alexander MacMillan, Montreal, Canada: My associates and I have had cases of traumatic enophthalmos caused by another type of injury, namely, skiing accidents, with which Dr. Pfeiffer has perhaps not come in contact. In some of these cases the floor of the orbit showed fracture, and I had felt that the enophthalmos was due to dropping of the globe into the antrum. However, a few years ago I saw a girl with enophthalmos, the result of a skiing accident, and yet no fracture of the orbit could be demonstrated roentgenographically. Dr. W. G. M. Byers was also interested in this subject at that time and took the question up with Dr. Whitnall, professor of anatomy at McGill University. Dr. Whitnall's explanation was that the suspensory ligament of Lockwood was probably ruptured. This ligament is formed from the sheaths of the inferior oblique and inferior rectus muscles, which join with the sheaths of the lateral and medial rectus muscles; they, in turn, are connected with the check ligaments, which are attached to the lateral and medial walls of the orbit. Since that time I have felt that rupture of this ligament is an important factor in the production of traumatic enophthalmos. In further support of this assumption, the superior maxilla may be removed and enophthalmos does not necessarily follow. I should like to know whether Dr. Pfeiffer agrees.

Dr. Peter C. Kronfeld, Chicago: I wonder whether Dr. Pfeiffer attaches any significance to the presence of preexisting gaps in the floor of the orbit. It is difficult to understand how an eyeball could transmit the amount of force necessary to fracture the intact floor of an orbit. Perhaps in some of these cases a congenital defect of the floor was present prior to the trauma. Such a defect could, perhaps, have been shown roentgenographically on the other side.

Dr. John H. Dunnington, New York: This study of Dr. Pfeiffer's has interested me very much, and I wish to cite a case of mine which bears out Dr. Pfeiffer's views. A girl came to me four years ago, shortly after having received a blow from a fist to her right eye. There were considerable swelling of the lids and other evidence of a severe contusion. A competent roentgenologist in New York, who specializes in sinus roentgenography, reported that there was no visible evidence of any fracture but the antrum was clouded. Within two weeks the girl had enophthalmos. I did not see her again until last week, when she returned, still complaining of diplopia and recession of the eyeball. This time a roentgenographic examination by Dr. Pfeiffer revealed the type of internal fracture of the orbit he has described. Unless one is particularly careful in the interpretation in such cases, the fracture is overlooked. I agree with Dr. Pfeiffer that a

fracture was demonstrable in all my cases of traumatic enophthalmos.

The tremendous amount of sinking of the upper fold can be completely eliminated by simple elevation of the eye. I operated on the patient with the extreme enophthalmos which Dr. Pfeiffer demonstrated. At the time of operation, simple elevation of the orbital contents from the antrum completely eliminated the sinking of the lid and the enophthalmos; I am convinced therefore that the deformity in cases of this kind is due entirely to the displacement of the tissues into the antrum. I do not mean to give the impression that this patient was permanently cured by operation, for sufficient time has not elapsed for one to judge the final result. The operative procedure used consisted of implantation of a piece of fat from the abdominal wall in the cavity created by elevation of the orbital contents. Decided improvement in the appearance was still present when the patient was last seen, two months after the operation.

Dr. C. A. Clapp, Baltimore: Dr. Pfeiffer stated that if the floor was sufficiently ruptured the eyeball might drop into the antrum, so that it would be invisible. Is this his deduction, or has he seen such a case?

Dr. Raymond L. Pfeiffer, New York: The success of roentgenography in the diagnosis of ophthalmic disturbances depends on the interest of the roentgenologist in studying the bones of the face and the orbit. The fine anatomic detail of the orbit which can be brought out in carefully prepared roentgenograms is not usually appreciated by the general roentgenologist. Recently a roentgenologist wrote that it was impossible to demonstrate the floor of the orbit in roentgenograms. This statement suggests why these fractures or deformities are not usually recognized.

I do not believe that rupture of Lockwood's ligament itself is sufficient to account for enophthalmos. Rupture of the ligament probably occurs with the fracture.

In answer to Dr. Kronfeld's question, I have never seen a congenital defect in the floor of the orbit in a roentgenogram. In the study of several large collections of dried skulls, I do not recall ever having seen any congenital defect or dehiscence of the floor. Whitnall mentioned hiatuses of the nasal walls.

In reply to Dr. Clapp's question, I have never seen an eye completely disappear from sight as a result of injury. In the next to the last case reported, the eye nearly disappeared from view. In this case the antrum was large. Occasionally one sees very large paranasal sinuses, certainly adequate to contain an eye and all the orbital contents. In the literature several cases have been recorded; von Becker and Tweedy both reported cases in which the eye disappeared from sight. In both instances the injury was produced by a cow's horn.

Arteritis of the Temporal Vessels: Report of a Case with Ocular Study. Dr. Lawrence T. Post and Dr. T. E. Sanders (by invitation), St. Louis.

Arteritis of the temporal vessels is a clinical entity, usually affecting elderly persons. It is characterized by thickening of the temporal arteries, which are painful and tender. There are also many painful areas in the scalp. It has some associated systemic symptoms, such as general malaise, fever, anorexia and anemia.

A number of cases have been reported in the general literature, in some of which ophthalmic studies were done. Except for an article from the Mayo Clinic (Johnson, R. H.; Harley, R. D., and Horton, B. I.: *Am. J. Ophth.* **26:** 147 [Feb.] 1943), there is no paper dealing primarily with the ocular lesions.

In the present case, a woman whose condition was one of the first reported on, about eight years ago, had a local cure at that time through surgical removal of the temporal arteries. She had no particular ocular complaints, but a peculiar lesion of the fundus was noted on routine examination.

Pigment Freckles of the Iris Associated with Malignant Melanoma of the Uvea. Dr. Algernon B. Reese, New York.

Localized accumulations of melanoblasts on the anterior surface of the iris are frequently noted in microscopic sections of eyes harboring a malignant melanoma of the uveal tract. These small multiple benign melanomas are referred to clinically as "freckles on the iris." The lesions have been interpreted as (1) coincidental, (2) implantation growths, (3) metastases, (4) extensions from the main lesion or (5) manifestations of multiple origin.

The present study indicates that the freckles are of multiple origin. In the cases reported the main lesion was in the choroid and the lesser manifestation on the iris. There occurred instances, however, in which the main lesion was in the iris and/or the ciliary body and the lesser lesion in the choroid, or in which the main lesion was in the choroid and the lesser manifestation elsewhere in the choroid. The freckles were composed of more matured cells than those comprising the main tumor and therefore were benign, with little or no power of active growth.

The clinical significance of pigment freckles on the iris lies in the fact that when they are noted in an eye in which a malignant melanoma is suspected and are not present, or are present

to a lesser degree, on the iris of the fellow eye, they offer substantiating evidence that the suspected lesion is a malignant melanoma. Also, the presence of pigment freckles on the iris seems to indicate active or malignant growth of the main lesion.

DISCUSSION

DR. F. H. VERHOEFF, Boston: It would be important to determine whether the freckles antedated the tumor of the choroid. Malignant melanoma may be neurogenic, as Dr. Reese mentioned, but I do not believe that Dr. Georgianna Theobald has proved it to be true. In a fully developed malignant melanoma of the choroid she could easily find nerves, whatever its origin may have been. There is a possible explanation of the freckles associated with malignant melanoma not mentioned by Dr. Reese. This is suggested by a frequent observation which I have made, namely, that a malignant melanoma of the corneal limbus produces an increase in the chromatophores in the tissue of the limbus for a considerable distance from the tumor. It would seem that from the tumor a substance is diffused which stimulates the chromatophores at a distance from it. In a case of a highly pigmented melanoma of the corneal limbus, with an implanted nonpigmented growth in the lower lid, I noted that the skin in a wide area around the implanted growth had become highly pigmented. This case seemed to prove that even an unpigmented malignant melanoma can stimulate the chromatophores around it.

DR. THEODORE L. TERRY, Boston: At birth the iris is not well developed, and the adult pigment characteristics are not evident. This change in pigmentation, or at least the appearance of pigmentation in the iris, is certainly not the development of freckles which Dr. Reese describes as occurring several years later. I wonder whether he has been able to identify any such groups of cells which might be regarded as an unpigmented freckle.

Some freckles in the iris in his illustrations appear to extend deep into the stroma, perhaps involving the muscle. Has he noticed any local alteration in the motility of the iris in that region? I mention this because I have always understood this alteration of motility to be strong presumptive evidence that the lesion is a malignant melanotic neoplasm of the iris. This sign of altered motility is demonstrated graphically by use, first, of miotics and, later, of mydriatics.

It is my impression that when malignant melanoma of the choroid is well enough advanced to invade the retina, it might free tumor cells in the eye, even producing implantations in the iris. Certainly, such implantations are to be seen in cases of retinoblastoma. Thus, it is difficult to dismiss completely the evidence of the possibility of implantation here; yet such an origin does not seem probable in the cases reported by Dr. Reese. In an eye removed because of melanoma of the iris, there was a melanoma not only in the iris but in the ciliary body; some distance away there was a mass of melanoma, consisting perhaps of not more than one hundred cells, resembling closely the tumor cells of the larger melanoma of the iris. Although this mass in the ciliary body represents a metastasis, most such tumors of the iris are not extremely malignant and rarely give rise to metastases, especially within the eye itself, as melanoma of the iris appears to be related to the melanoma of the uveal tract approximately to the same degree to which the basal cell carcinoma is related to the really malignant epidermoid carcinoma. The lesion in the ciliary body may, in turn, be a secondary lesion stimulated by the primary lesion in the iris, if Dr. Reese is correct in his assumption that the freckles of the iris are associated with a more posterior uveal melanoma. It was my belief that the immaturity of the cells in each region was of approximately the same grade. I shall send one of these sections to Dr. Reese and see what he can make out of it.

Another point to be considered is the possible relation of this disease to melanosis oculi. At one time, Doherty pointed out that of persons who had melanosis oculi, approximately 25 per cent later showed malignant changes in the uveal tissue. The relation of freckles to malignant melanoma of the posterior uvea may indeed be similar to that of melanosis oculi to malignant uveal disease, the one being multiple and the other diffuse. One often sees a conspicuous overproduction of retinal pigment epithelium adjacent to malignant melanoma of the choroid, but heretofore this has been thought to be a result of irritation, whereas it might be related to the lesion that Dr. Reese has presented.

DR. ARNOLD KNAPP, New York: In confirmation of Dr. Reese's statement that this condition cannot be a process of implantation on the surface of the iris, I shall mention briefly the case of a woman aged 32 who was referred to me with the diagnosis of choroiditis and glaucoma. Trephining with iridectomy had been done, and she presented the usual coloboma. The color of the iris was almost inky black. A tumor could easily be seen behind one pillar of the coloboma. The trephine opening was functioning, and the raised epithelium was studded with very small spots. The eye was removed, and microscopic examination showed that a tumor of the ciliary body had broken through at its apex and that the surface of the iris was plastered with metastatic tumors, which had also invaded the angle of the iris; the conjunctival spots were also metastases, according to Dr. James Ewing.

DR. ALGERNON B. REESE, New York: Dr. Verhoeff objected to my reference to the neurogenic nature of malignant melanoma of the uvea. However, it will be recalled that I prefaced this comment by saying that I assumed that Dr. Theobald's contention was correct.

Dr. Verhoeff's idea that some substance may emanate from the main lesion and produce these pigment lesions elsewhere seems plausible.

The question which Dr. Terry asked regarding the possible occurrence of unpigmented freckles of the iris is interesting. This possibility had occurred to me, and I intend to watch for them. In the past even the pigmented ones have been overlooked. Dr. Terry asked whether benign melanoma of the iris caused any alteration in motility. I cannot answer this question specifically, but I judge that they do not, unless the lesion involves more than the anterior surface of the iris. In 1 of the cases shown the lesion involved the entire thickness of the iris and produced ectropion of the uvea. Such a lesion, I feel sure, altered the motility of the iris.

Pathologic Changes in the Lens Associated with Nontraumatic Iritis. DR. BERNARD SAMUELS, New York.

This article was published in full in the January 1944 issue of the ARCHIVES, page 8.

Less Evident Causes of Lowered Acuity in Senile Persons. DR. RALPH I. LLOYD, Brooklyn.

It is usually possible to find in the eye of every one over 60 some cause of lowered function. Glaucoma, cataract and the vascular changes in the retinal vessels, with well known chorioretinal lesions, explain most of the beginning failures of vision in older patients. The less evident conditions may be operating on the cornea from its posterior surface or are faintly seen in the macular area as edema of the retina and choriocapillaris; or the eye may seem normal but examination of the visual fields may reveal progressive lesions, due to vascular degeneration and pressure on the optic chiasm or the optic nerve in the optic canal. This last condition has been especially studied by Fuchs and Liebrecht. Lesions in the cuneus producing hemianopsia have been better studied than the conditions first mentioned.

DISCUSSION

DR. JONAS S. FRIEDENWALD, Baltimore: I disagree with Dr. Lloyd in regard to a number of the interpretations he made in the early part of his paper. He spoke of deposits of fibrin on the back of the cornea in his cases. I should like to know how he knows it is fibrin.

He said the relation of endothelial dystrophy to Fuchs's dystrophy was first shown by Vogt. It had been my impression that this relation was first called to attention by my father; at least, at the time my father published his paper, I was unable to find any reference to Vogt on that subject.

Dr. Lloyd said that improvement in vision in the early stage of Fuchs's dystrophy during the day and its reduction at night are due to massage of the eyeball during the day. I believe Cogan's important contribution to the mechanism of corneal edema in Fuchs's dystrophy and the role of drying of the corneal epithelium by exposure deserve consideration.

DR. RALPH I. LLOYD, Brooklyn: Dr. Friedenwald asks how I know the fragments on the posterior surface of the cornea in the illustrations are fibrin. I do not know from my own examination; but my instruction in slit lamp study was under Koeppe, and similar deposits then seen were said to be fibrin. That the change in acuity in cases of Fuchs's dystrophy results from massage of the edematous anterior surface of the cornea during winking is my own opinion.

Recurring Attacks of Concomitant Exotropia, Each Followed by Transient Esotropia: Migraine the Probable Cause. DR. F. H. VERHOEFF, Boston.

This article was published in full in the December 1943 issue of the ARCHIVES, page 727.

DISCUSSION

DR. WALTER B. LANCASTER, Boston: The relation of accommodation and convergence is even more important than most persons realize. I shall report a case which closely resembles Dr. Verhoeff's. A student nurse had difficulty in carrying on her studies. Examination showed hypermetropia of 1.25 D. with a small cylinder; vision was 6/5 in each eye. Tests with the Maddox rod showed from 12 to 16 prism diopters of esophoria. It is characteristic of cases of this condition that the amount of esophoria is variable, suggestive of its dependence on a spastic condition. When she was tested with the Maddox tangent cross (the Bielschowsky method with the vertical prism to produce dissociation and a red glass before one eye to identify the image), she indicated the position of the red image, but when she was asked to give the number on the scale, she said "I cannot see the numbers." She required a — 2 or 3 D. sphere to see the numbers. Obviously, she was accommodating and thus producing an esophoria which showed on the Maddox scale as 10, 12 or 16 prism diopters. With the cover test, without presentation of objects to stimulate her accommodation, simply with the light, she showed an exophoria of 6 prism diopters. Obviously, then, the underlying condition was exophoria, with the abnormal esophoria superimposed by the spasm of accom-

modation. This case differed from Dr. Verhoeff's in that the condition was apparent at every visit. It was not a matter of periodic attacks. With suitable treatment, she was able to carry on her work as a student nurse and complete her course. One of my first thoughts was operation, but, fortunately, I waited long enough to satisfy myself that it was not the solution.

As to how the abnormal esophoria is induced, it suffices at present to say that it is fundamentally due to the close relation between accommodation and convergence, and when the regulation of that relation breaks down, when for one cause or another the patient accommodates more than he ought, esophoria is present.

Some of the patients who were treated by the Bates method come under this heading. If a patient has esophoria and he can be taught to look at distant objects without accommodating, his eyes are straight; conversely, if distant objects are blurred, and he can be induced to converge his eyes, he overcomes his hypermetropia and gets clear vision; but to keep that up and have efficient sight is another matter, and failure is a source of disastrous accidents.

DR. THOMAS D. ALLEN, Chicago: Dr. Verhoeff mentioned a personal, as well as a family, history of migraine in his case. He also noted angioneurotic edema, which is associated with migraine. I wonder whether allergy may have been a cause of the migraine and the angioneurotic edema. I wonder whether Dr. Verhoeff has made a histamine test in this case and whether there was any notable eosinophilia.

DR. F. H. VERHOEFF, Boston: Dr. Lancaster's interesting comments emphasized the difficulty of measuring heterophoria but did not seem to throw any light on my case. In reply to Dr. Allen, I may say that the cause of the migraine was a separate problem, which I did not discuss and which, as a matter of fact, I did not solve. Ergotamine was not used until the end of the last attack. I planned to try histamine and, if necessary, other drugs if another series of attacks should begin.

Keratectomies for Treatment of Corneal Opacities. DR. RAMÓN CASTROVIEJO, New York.

This article was published in full, with discussion, in the July 1944 issue of the ARCHIVES, page 11.

Recession of the Trochlea. DR. WENDELL L. HUGHES, Hempstead, N. Y.

This procedure is devised to reduce the action of the superior oblique muscle when necessary. The usual indications are overaction of the muscle, whether primary or secondary to paralysis or paresis of the associated inferior rectus muscle of the opposite eye. Two cases in which this procedure was performed have previously been reported. The experience with 4 more cases is here presented.

The technic is simple and can be carried out under local or general anesthesia.

A cutaneous incision is made parallel with the orbital margin in its upper nasal position, in the region of the trochlea, nasal to the supraorbital notch. The incision, 1 to 1.5 cm. long, is carried down to the bone through the periosteum on the orbital rim. The periosteum, which is densely adherent to the bone in the area of the trochlea, is turned backward along the roof of the orbit about 1.5 cm. Two parallel incisions at right angles to the first incision are made in the periosteum to which the trochlea is attached. The trochlea is easily pushed posteriorly by means of a pressure dressing. The skin is closed and a small roll of gauze placed over the area of the trochlea. This is held in place, the trochlea being pushed posteriorly by means of a pressure dressing. In each of the 6 cases the result has been a correction of the downshoot in the nasal field, due to the overaction of the superior oblique muscle.

DISCUSSION

DR. WENDELL L. HUGHES, Hampstead, N. Y.: The superior oblique muscle itself is an irregularly overacting muscle in cases of this type; it is a spastic muscle, and no accurate and consistent measurements are possible. The hypotropia varies with different positions of gaze, being greatest when the eye is rotated nasally and down by the action of the medial rectus and superior oblique muscles. The purpose is to reduce the depressing effect of this muscle where the depressing effect of the superior oblique muscle is most marked and where the deviation of the eye is greatest.

No attempt is made to put the trochlea back so many millimeters. The procedure is comparable to section of the inferior oblique muscle for spasm.

Choice of the Fixating Eye in Paralytic and Nonparalytic Strabismus. DR. JAMES W. WHITE, New York.

While strabismus, either paralytic or nonparalytic, may be alternating for distant and near vision, it may be seen as right or left convergent or divergent strabismus or as vertical strabismus. The same eye may be the fixating eye for distant and near vision, or one eye may fixate for distance, while the fellow eye may fixate for near vision. Often, when one eye fixates for both distant and near vision, fixation may change as the eyes are directed toward the six cardinal fields. This choice of fixation may result in more or less variation in the amount of deviation and may,

and often does, change the treatment radically, especially in the surgical correction of the strabismus. Cases are cited.

Tuberous Sclerosis: Report of a Case. Dr. Ernest F. Krug, New York.

This article was published in full in the January 1944 issue of the Archives, page 68.

DISCUSSION

Dr. Walter I. Lillie, Philadelphia: The case of tuberous sclerosis presented is classic; the disease is characterized by the existence of hard tumefactions of the cerebral convolutions, formed of aggregations of a peculiar cell, related both to glial and to ganglion cells.

I wish to emphasize three features of this case: First, the patient had a history of increased intracranial pressure associated with headache and choked disk, but without localizing signs of the lesion; in such a case a complete general and neurologic examination should be made as soon as possible. Second, although the roentgenograms showed evidence of increased intracranial pressure, with associated changes in the sella turcica, the visual fields did not conform with those observed with a chiasmal lesion. This in itself is a manifestation that the change in the sella turcica is only another sign of increased intracranial pressure and has no localizing value. A tumor of the third ventricle is suprasellar but does not necessarily involve the chiasm unless the optic recess of the third ventricle is involved. The third, and most important, feature of the case was the progressive loss of vision associated with choking of the disk. If vision is to be preserved, treatment should be instituted before definite signs of secondary optic nerve atrophy are visible with the ophthalmoscope. Even though the tumor cannot be located accurately, in any case of choked disk occipital decompression should be done as soon as vision is diminished, for the beginning binasal hemianopsia is positive evidence of changes in the optic nerve fibers at the disk, due to continued pressure from the edema. If the intracranial pressure is not relieved, central visual acuity will be affected. Usually there is progressive loss of vision, even though the intracranial condition is relieved later.

At present neurosurgeons are able to remove space-taking lesions in almost any portion of the brain, with an extremely low mortality rate, and the earlier this procedure can be instituted, the better the end results will be.

Choked Disk and Low Intrathecal Pressure Associated with Tumor of the Brain. Dr. P. J. Leinfelder, Iowa City.

In a review of a number of proved cases of cerebral tumor, it was noted that under some circumstances choked disk occurred even though normal intrathecal pressure was recorded on lumbar puncture. A study of a series of 12 cases revealed that in spite of low intrathecal pressure, observation at operation or autopsy disclosed definite evidence of increased intracranial pressure. In only 2 cases was there a demonstrable block in the circulation of the cerebrospinal fluid. The records in these cases suggest that the lumbar puncture does not invariably give a true estimate of the intracranial pressure and that the choked disk is of more importance in the diagnosis of increased intracranial pressure.

DISCUSSION

Dr. Albert N. Lemoine, Kansas City, Mo.: Did Dr. Leinfelder record the intraocular pressure in these cases?

Dr. Walter I. Lillie, Philadelphia: Dr. Leinfelder's presentation brings out a point which is overlooked clinically. Many remember a paper presented before this society by Dr. A. J. Bedell (Papilledema Without Increased Intracranial Pressure, *Tr. Am. Ophth. Soc.* **39:** 242, 1941; Arch. Ophth. **27:** 810 [April] 1942), in which all the cases cited were classic examples of acute optic neuritis in that precipitous loss of central visual acuity was associated with changes in the central visual fields. In Dr. Leinfelder's cases choking of the disks was not accompanied by any alteration in the central visual acuity or changes in the visual field. Papilledema is a sign, not a disease entity, and is associated with many ocular syndromes. The occurrence of normal spinal fluid pressure with the presence of a proved intracranial space-taking lesion is not uncommon, and the absence of increase in spinal fluid pressure does not rule out the presence of tumor. Usually a tumor of the posterior fossa produces signs of great pressure early in the course of the syndrome, atlhough in a certain percentage of cases there is no change in the readings of the cerebrospinal fluid pressure.

Dr. Leinfelder has raised the question of the danger of examination of the spinal fluid in cases of tumor of the posterior fossa. In my experience, the risk in making such an examination has been present in cases of the supratentorial, rather than of the infratentorial, type, as the danger lies in the herniation of the cerebrum through the tentorium rather than in herniation of the medulla.

The occurrence of visual changes associated with choked disk designates a pathologic condition of the disk, due to the long-standing edema, and has no localizing value as to the position of the intracranial lesion. The occurrence of nasal contraction in the visual fields is evidence of beginning secondary optic nerve atrophy, even if pallor of the optic disk cannot be observed with the ophthalmoscope because of the edema. As soon as the binasal defect of the visual field

appears in a case with choking of the disk, the increased intracranial pressure should be relieved, even though the tumor cannot be removed, for if the pressure is allowed to continue, progressive secondary optic nerve atrophy will take place, with resulting loss of vision.

Dr. P. J. Leinfelder, Iowa City: Dr. Lemoine asked about the intraocular pressure in these cases. Unfortunately, tonometric readings were not made. However, in every case in which the patient had low intrathecal pressure and choked disk I always took the tension with my fingers, and in no instance was the eyeball soft. I do not believe that intraocular pressure played a part in these cases.

Dr. Lillie asked about the cause. The etiologic factor in each case was proved histologically, and in some instances pathologic specimens were obtained.

Herniation of the medulla after lumbar puncture has long been a subject of discussion by neurosurgeons and neurologists. I take no particular stand on the question in the presentation of this paper.

Book Reviews

The Romance of Medicine. By Benjamin Lee Gordon, M.D. Price, $5. Pp. 624, with 146 illustrations. Philadelphia: F. A. Davis Company, 1944.

This is an interesting book for both the medical man and the layman, in the writing of which the author has evidently enjoyed himself to the full. From it one may learn much about "magicians, astrologers, necromancers, clairvoyants, faith healers, and fortune tellers" and of "superstitions, folklore, legend, fancies, fables, and facts"—to borrow phrases of the author. Not only are there chapters on such subjects as "early ideas of gestation and genetics," "the secret of the human automaton," "the location of the vital energy," "demonology and hysteria," "demonology and bacteriology," "astrology and medicine," "the power of mystic symbols and magical gestures," "the scapegoats of medicine" and "signature and other healing concepts," but other chapters discuss "Hebrew and Christian ideas of the after-life" and "speculative modern concepts." Dr. Gordon also provides an excellent list of diseases which bear the names of saints.

Example after example is given of the various beliefs held among ancient peoples or savage tribes of today about the nature or causation of disease—examples drawn from all parts of the world. There are many quotations from "The Golden Bough," by Sir James Frazer, and numerous references to the literature of the Old Testament and to the Talmud. So it is a book of reference, made handy by means of a good index, but after reading the work one has to confess so many instances have been cited that they become confused in one's mind. One has often wondered how savages dared to take even one trembling step for fear that they might, in so doing, incur the wrath of the spirits which animated every object of nature about them.

Dr. Gordon expresses his view thus:

> . . . With regard to psychotherapy, the occult healer again anticipated the modern psychiatrist. Psychotherapy, as it will be shown, was a development of occult healing, just as bacteriology was an outgrowth of demonology, astronomy a derivative of astrology, and chemistry an amplification of alchemy.

It is sobering to think that today astrology has so many followers; Dr. Gordon quotes from an article, "Health and Your Horoscope," which was published by a medical astrologer in 1940.

The author is an ophthalmologist, and his articles on the history of ophthalmology are well known. For example, "The Problem of the Crystalline Lens" and "Oculists and Occultists" were published in the ARCHIVES (14: 774 [No.] 1935 and 22: 25 [July] 1939). Chapter 12 deals with that widespread superstition the "evil eye" and the part it has played in the lives of many people. That a visual spirit dwelt within the eye and other such ideas are also discussed. The author mentions numerous curious remedies employed in the treatment of diseases of the eye. Is one to understand from the illustration on page 67, described as an "eighth century drawing of the eyeball . . . (from the author's collection)," that he possesses an original drawing dating from that century, or does he have merely a reproduction of such a drawing?

Almost every plant and parts of almost every animal have been tried as remedies in the treatment of symptoms or in the attempt to cure disease, and are being used in some part of the world today. Is it merely a case of trial and error? The book has yet to be written which traces the history of remedies of proved worth. And, in discussing any of these drugs, one must bear in mind that it is difficult to rule out faith, which is still so powerful a factor in recovery. The imponderabilia should be pondered.

The author discusses "the King's touch" in the treatment of scrofula—in fact, the subject is taken up twice (pages 249 to 250 and 358 to 359), and two paragraphs are repeated. One learns twice how Dr. Samuel Johnson was "touched" by Queen Anne. This, and other errors, might well be corrected when a new edition of the work is called for.

William Withering learned the use of foxglove (digitalis) from an old Shropshire woman—but surely her story is overdramatized! Remedies for scurvy were in use about two hundred years before James Lind wrote of the advantages of oranges, limes and lemons, but was it not more likely "spruce beer" than a decoction of sassafras which the sailors of Jacques Cartier employed?

The book has many illustrations, but they might be much better. Many of them are merely fanciful creations of today. And why, when adequate photographs or engraved portraits could easily have been found, was it necessary to use modern redrawings of them? There are many mistakes in the spelling of proper names, such as Frazier, Hyrtle, Jonathan Right, Levator, John Astruk, Finicus, Ashmoli, John of Gladdesden, St. Hildegard of Bengen, Calumella, Menelous and Lavaran, and 1820 should be 1880. Marlowe, not Shakespeare, was the author of "The Jew of Malta."

The keynote of the book is sounded when Dr. Gordon says: "Modern medicine is like a fruit tree of a well-cultivated grove or orchard whose remote origin may be traced to the wild tree of the forest."

Blood Supply of the Visual Pathway. By Calvin M. Kershner, M.D., M.S., in Ophthalmology. Price $3. Pp. 160, with 33 illustrations. Boston: Meador Publishing Company, 1943.

In the consideration of the structure of the visual pathways the blood supply is of great importance. This monograph has gathered together in a systematic way what is known about this subject. The general scheme of the blood supply to the visual pathways is first given; then each division of the pathway which has a distinctive blood supply is described in detail. For convenience of discussion the visual pathway is divided into an anterior and a posterior section. The anterior section comprises the orbital portion and the optic nerve up to the chiasm; the posterior section includes the rest of the visual pathway, composed of the chiasm, the tracts, the external geniculate bodies, the radiation of Gratiolet and the visual cortical areas in the occipital region. The two parts are not strictly separate, as the blood supply of the chiasm is related to both parts. After a description of the four neurons, the blood supply of the anterior section is traced from the aorta by way of the common carotid artery and its main branches, and particularly by one of the terminals, the very important ophthalmic artery. The venous drainage of the orbit and the venous anastomoses come next for consideration: the facial vein, the pterygoid plexus and the intracranial sinuses. The blood for the posterior portion comes from the internal carotid and the vertebral arteries, and the venous drainage is the same as that of the midbrain and the posterior cortical area, where the blood finally enters the sinuses of the dura mater. The second, and most important, part of the book takes up (*a*) the retinal elements, with a detailed description of the retinal and choroidal circulation; (*b*) the optic nerve, and (*c*) the lateral geniculate body, the optic radiations and the cortex—the area striata. In this section the relation of the blood supply to clinical and pathologic conditions is also mentioned and their importance in the understanding of clinical symptoms demonstrated. The text is clearly written, and the subject matter is illustrated by simple drawings in black and white, taken from recognized authorities. The book concludes with a complete bibliography.

The author has succeeded, with commendable industry, in providing a valuable monograph which contains in a relatively small space much important, and often not very accessible, information, which will be found useful by the ophthalmologist and the neurologist.

ARNOLD KNAPP.

Minor Surgery. Edited by Humphry Rolleston and Alan Moncrieff. Price, $5. Pp. 174. New York: Philosophical Library, Inc., 1943.

The subject of minor surgery is treated in 18 chapters and covers practically all minor surgical conditions, such as minor wounds, sprains, disabilities of the feet and hands, and minor disturbances of the nose and throat, ear, eye, skin and genitourinary system, and anesthesia. These chapters are written by prominent English surgeons. The subject matter is covered in 155 pages, with an index of 18 pages.

The chapter on the eye is written by J. S. Arkle, ophthalmic surgeon to the Royal Victoria Infirmary, Newcastle-upon-Tyne. In this chapter the following subjects are discussed: removal of foreign bodies; mild infections, such as stys; cysts; dacryocystitis; repair of laceration of the lids, and treatment of burns of the eye. In the small space (6 pages) that this chapter occupies, only a superficial description of the procedures can, of course, be given.

ARNOLD KNAPP.

Directory of Ophthalmologic Societies *

INTERNATIONAL

International Association for Prevention of Blindness

President: Dr. P. Bailliart, 66 Boulevard Saint-Michel, Paris, 6e, France.
Secretary-General: Prof. M. Van Duyse, Université de Gand, Gand, Prov. Ostflandern, Belgium.
All correspondence should be addressed to the Secretariat, 66 Boulevard Saint-Michel, Paris, 6e, France.

International Ophthalmologic Congress

President: Prof. Nordenson, Serafimerlasarettet, Stockholm, Sweden.
Secretary: Dr. Ehlers, Jerbanenegade 41, Copenhagen, Denmark.

International Organization Against Trachoma

President: Dr. A. F. MacCallan, 17 Horseferry Rd., London, England.

Pan-American Congress of Ophthalmology

President: Dr. Harry S. Gradle, 58 E. Washington St., Chicago.
Executive Secretaries: Dr. Conrad Berens, 35 E. 70th St., New York. Dr. M. E. Alvaro, 1511 Rua Consolacão, São Paulo, Brazil.

FOREIGN

All-India Ophthalmological Society

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Secretary: Dr. G. Zachariah, Flitcham, Marshall's Rd., Madras.

British Medical Association, Section on Ophthalmology

President: Dr. W. Clark Souter, 9 Albyn Pl., Aberdeen, Scotland.
Secretary: Dr. Frederick Ridley, 12 Wimpole St., London, W. 1.

Chengtu Ophthalmological Society

President: Dr. Eugene Chan.
Secretary: Dr. K. S. Sun.
Place: Eye, Ear, Nose and Throat Hospital, Chengtu, China.

Chinese Ophthalmology Society

President: Dr. C. H. Chou, 363 Avenue Haig, Shanghai.
Secretary: Dr. F. S. Tsang, 221 Foochow Rd., Shanghai.

Chinese Ophthalmological Society of Peiping

President: Dr. H. T. Pi, Peiping Union Medical College, Peiping.
Secretary: Dr. C. K. Lin, 180 Hsi-Lo-yen Chienmeng, Peiping.
Place: Peiping Union Medical College, Peiping. Time: Last Friday of each month.

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Hungarian Ophthalmological Society

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Assistant Secretary: Dr. Stephen de Grósz, University Eye Hospital, Máriautca 39, Budapest.
All correspondence should be addressed to the Assistant Secretary.

Midland Ophthalmological Society

President: Dr. W. Niccol, 4 College Green, Gloucester, England.
Secretary: Mr. T. Harrison Butler, 61 Newhall St., Birmingham 3, England.
Place: Birmingham and Midland Eye Hospital.

North of England Ophthalmological Society

President: Dr. A. MacRae, 6 Jesmond Rd., Newcastle-upon-Tyne, England.
Secretary: Dr. Percival J. Hay, 350 Glossop Rd., Sheffield 10, England.
Place: Manchester, Bradford, Leeds, Newcastle-upon-Tyne, Liverpool and Sheffield, in rotation. Time: October to April.

Ophthalmological Society of Australia

President: Dr. A. James Flynn, 135 Macquarie St., Sydney.
Secretary: Dr. D. Williams, 193 Macquarie St., Sydney.

Ophthalmological Society of Egypt

President: Prof. Dr. Mohammed Mahfouz Bey, Government Hospital, Alexandria.
Secretary: Dr. Mohammed Khalil, 4 Baehler St., Cairo.
All correspondence should be addressed to the secretary, Dr. Mohammed Khalil.

Ophthalmological Society of the United Kingdom

President: Mr. Charles B. Goulden, O.B.E., M.D., M.Ch., 89 Harley St., London, England.
Secretary: Mr. Frank W. Law, M.A., M.D., F.R.C.S., 30 Devonshire Pl., London W. 1, England.

Ophthalmology Society of Bombay

President: Dr. D. D. Sathaye, 127 Girgaum Rd., Bombay 4, India.
Secretary: Dr. H. D. Dastur, Dadar, Bombay 14, India.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

Oxford Ophthalmological Congress

Master: Mr. P. G. Doyne, 60 Queen Anne St., London, W. 1, England.
Secretary-Treasurer: Dr. F. A. Anderson, 12 St. John's Hill, Shrewsbury, England.
Place: Oxford, England. Time: July 8-9, 1943.

Palestine Ophthalmological Society

President: Dr. Arieh Feigenbaum, Abyssinian St. 15, Jerusalem.
Secretary: Dr. E. Sinai, Tel Aviv.

Polish Ophthalmological Society

President: Dr. W. Kapuściński, 2 Waly Batorego, Poznań.
Secretary: Dr. J. Sobański, Lindley'a 4, Warsaw.
Place: Lindley'a 4, Warsaw.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and keep it up to date.

ROYAL SOCIETY OF MEDICINE, SECTION OF OPHTHALMOLOGY

President: Col. F. A. Juler, 96 Harley St., London, W. 1, England.
Secretary: Dr. Harold Ridley, 60 Queen Anne St., London, W. 1, England.

SÃO PAULO SOCIETY OF OPHTHALMOLOGY

President: Dr. W. Belfort Mattos, Caixa Postal, 4086, São Paulo, Brazil.
Secretary: Dr. Silvio de Almeida Toledo, Enfermaria Santa Luzia, Santa Casa de Misericordia, Cesario Motta, St. 112, São Paulo, Brazil.

SOCIEDAD ARGENTINA DE OFTALMOLOGIA

Chairman: Dr. Jorge Malbrán, Buenos Aires.
Secretary: Dr. Benito Just Tiscornia, Santa Fe 1171, Buenos Aires.

SOCIEDAD OFTALMOLOGIA DEL LITORAL, ROSARIO (ARGENTINA)

President: Prof. Dr. Carlos Weskamp, Laprida 1159, Rosario.
Secretary: Dr. Juan M. Vila Ortiz, Córdoba 1433, Rosario.
Place: Rosario. Time: Last Saturday of every month, April to November, inclusive. All correspondence should be addressed to the President.

SOCIEDADE DE OPHTHALMOLOGIA E OTO-RHINO-LARYNGOLOGIA DA BAHIA

President: Dr. Theonilo Amorim, Barra Avenida, Bahia, Brazil.
Secretary: Dr. Adroaldo de Alencar, Brazil.
All correspondence should be addressed to the President.

SOCIETÀ OFTALMOLOGICA ITALIANA

President: Prof. Dott. Giuseppe Ovio, Ophthalmological Clinic, University of Rome, Rome.
Secretary: Prof. Dott. Epimaco Leonardi, Via del Gianicolo, 1, Rome.

SOCIÉTÉ FRANÇAISE D'OPHTALMOLOGIE

Secretary: Dr. René Onfray, 6 Avenue de la Motte Picquet, Paris, 7e.

SOCIETY OF SWEDISH OPHTHALMOLOGISTS

President: Prof. K. G. Ploman, Stockholm.
Secretary: Dr. K. O. Granström, Södermalmstorg 4 III tr., Stockholm, Sö.

TEL AVIV OPHTHALMOLOGICAL SOCIETY

President: Dr. D. Arieh-Friedman, 96 Allenby St., Tel Aviv, Palestine.
Secretary: Dr. Sadger Max, 9 Bialik St., Tel Aviv, Palestine.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Frederick C. Cordes, 384 Post St., San Francisco.
Secretary: Dr. R. J. Masters, 23 E. Ohio St., Indianapolis.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY, SECTION ON OPHTHALMOLOGY

President: Dr. Lawrence T. Post, Metropolitan Bldg., St. Louis.
President-Elect: Dr. Gordon B. New, Mayo Clinic, Rochester, Minn.
Executive Secretary-Treasurer: Dr. William L. Benedict, 101–1st Ave. Bldg., Rochester, Minn.

AMERICAN OPHTHALMOLOGICAL SOCIETY

President: Dr. S. Judd Beach, 704 Congress St., Portland, Maine.
Secretary-Treasurer: Dr. Walter S. Atkinson, 129 Clinton St., Watertown, N. Y.

ASSOCIATION FOR RESEARCH IN OPHTHALMOLOGY, INC.

Chairman: Dr. Frederick C. Cordes, 384 Post St., San Francisco.
Secretary-Treasurer: Major Brittain F. Payne, School of Aviation Medicine, Randolph Field, Texas.

CANADIAN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY

President: Dr. Alexander E. MacDonald, 170 St. George St., Toronto.
Secretary-Treasurer: Dr. L. J. Sebert, 170 St. George St., Toronto.

CANADIAN OPHTHALMOLOGICAL SOCIETY

President: Dr. R. Evatt Mathers, 34½ Morris St., Halifax, N. S.
Secretary-Treasurer: Dr. Kenneth B. Johnston, Suite 1, 1509 Sherbrooke St. W., Montreal.

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS

President: Mr. Mason H. Bigelow, 1790 Broadway, New York.
Secretary: Miss Regina E. Schneider, 1790 Broadway, New York.
Executive Director: Mrs. Eleanor Brown Merrill, 1790 Broadway, New York.

SECTIONAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. N. Zwaifler, 46 Wilbur Ave., Newark.
Secretary: Dr. William F. Keim Jr., 25 Roseville Ave., Newark.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

CENTRAL WISCONSIN SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. L. J. Friend, 425 E. Grand Ave., Beloit, Wis.
Secretary: Dr. G. L. McCormick, 626 S. Central Ave., Marshfield.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. Paul A. Chandler, 5 Bay State Rd., Boston.
Secretary-Treasurer: Dr. Merrill J. King, 264 Beacon St., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. D. H. O'Rourke, 1612 Tremont Pl., Denver.
Secretary-Treasurer: Dr. C. Allen Dickey, 450 Sutter St., San Francisco.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. L. L. Bull, 1215–14th Ave., Seattle, Wash.
Secretary-Treasurer: Dr. Barton E. Peden, 301 Stimson Bldg., Seattle 1.
Place: Seattle or Tacoma, Wash. Time: Third Tuesday of each month except June, July and August.

ROCK RIVER VALLEY EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. J. Sheldon Clark, 27 E. Stephenson St., Freeport, Ill.
Secretary-Treasurer: Dr. Harry R. Warner, 321 W. State St., Rockford, Ill.
Place: Rockford, Ill., or Janesville or Beloit, Wis. Time: Third Tuesday of each month from October to April, inclusive.

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. M. H. Pike, Midland, Mich.
Secretary-Treasurer: Dr. H. H. Heuser, 207 Devidson Bldg., Bay City, Mich.
Place: Saginaw or Bay City, Mich. Time: Second Tuesday of each month, except July, August and September.

SIOUX VALLEY EYE AND EAR ACADEMY

President: Dr. J. C. Decker, 515 Francis Bldg., Sioux City, Iowa.
Secretary-Treasurer: Dr. J. E. Dvorak, 408 Davidson Bldg., Sioux City, Iowa.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. John H. Burleson, 414 Navarro St., San Antonio, Texas.
Secretary: Dr. J. W. Jervey Jr., 101 Church St., Greenville, S. C.

SOUTHWESTERN ACADEMY OF EYE, EAR, NOSE AND THROAT

President: Dr. H. L. Brehmer, 221 W. Central Ave., Albuquerque, N. Mex.
Secretary: Dr. A. E. Cruthirds, 1011 Professional Bldg., Phoenix, Ariz.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. W. M. Dodge, 716 First National Bank Bldg., Battle Creek.
Secretary-Treasurer: Dr. Kenneth Lowe, 25 W. Michigan Ave., Battle Creek.
Time: Last Thursday of September, October, November, March, April and May.

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Ray Parker, 218 Franklin St., Johnston, Pa.
Secretary-Treasurer: Dr. J. McClure Tyson, Deposit Nat'l Bank Bldg., DuBois.

STATE

ARKANSAS STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. Raymond C. Cook, 701 Main St., Little Rock.
Secretary: Dr. K. W. Cosgrove, Urquhart Bldg., Little Rock.

COLORADO OPHTHALMOLOGICAL SOCIETY

President: Dr. C. A. Ringle, 912–9th Ave., Greeley.
Secretary: Dr. W. A. Ohmart, 1102 Republic Bldg., Denver.
Place: University Club, Denver. Time: 7:30 p. m., third Saturday of each month, October to May, inclusive.

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. F. L. Phillips, 405 Temple St., New Haven.
Secretary-Treasurer: Dr. W. H. Turnley, 1 Atlantic St., Stamford, Conn.

EYE, EAR, NOSE AND THROAT CLUB OF GEORGIA

President: William O. Martin Jr., Doctors Bldg., Atlanta.
Secretary-Treasurer: Dr. C. K. McLaughlin, 526 Walton St., Macon.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. F. McK. Ruby, Union City.
Secretary: Dr. Edwin W. Dyar Jr., 23 E. Ohio St., Indianapolis.
Place: French Lick. Time: First Wednesday in April.

IOWA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. J. K. Von Lackum, 117–3d St. S. E., Cedar Rapids.
Secretary-Treasurer: Dr. B. M. Merkel, 604 Locust St., Des Moines.

LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. Val H. Fuchs, 200 Carondelet St., New Orleans.
Secretary-Treasurer: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.

MICHIGAN STATE MEDICAL SOCIETY, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Robert H. Fraser, 25 W. Michigan Ave., Battle Creek.
Secretary: Dr. R. G. Laird, 114 Fulton St., Grand Rapids.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. George E. McGeary, 920 Medical Arts Bldg., Minneapolis.
Secretary: Dr. William A. Kennedy, 372 St. Peter St., St. Paul.
Time: Second Friday of each month from October to May.

Montana Academy of Oto-Ophthalmology

President: Dr. William Morrison, 208 N. Broadway, Billings, Mont.
Secretary: Dr. Fritz D. Hurd, 309 Medical Arts Bldg., Great Falls.

Nebraska Academy of Ophthalmology and Otolaryngology

President: Dr. W. Howard Morrison, 1500 Medical Arts Bldg., Omaha.
Secretary-Treasurer: Dr. John Peterson, 1307 N St., Lincoln.

New Jersey State Medical Society, Section on Ophthalmology, Otology and Rhinolaryngology

Chairman: Dr B. E. Failing, 31 Lincoln Park, Newark.
Secretary: Dr. George Meyer, 410 Haddon Ave., Camden.

New York State Medical Society, Eye, Ear, Nose and Throat Section

Chairman: Dr. Harold J. Joy, 504 State Tower Bldg., Syracuse 2.
Secretary: Dr. Maxwell D. Ryan, 660 Madison Ave., New York 21.

North Carolina Eye, Ear, Nose and Throat Society

President: Dr. Hugh C. Wolfe, 102 N. Elm St., Greensboro.
Secretary: Dr. Vanderbilt F. Couch, 104 W. 4th St., Winston-Salem.

North Dakota Academy of Ophthalmology and Oto-Laryngology

President: Dr. W. L. Diven, City National Bank Bldg., Bismarck.
Secretary-Treasurer: Dr. A. E. Spear, 20 W. Villard, Dickenson.

Oregon Academy of Ophthalmology and Oto-Laryngology

President: Dr. Paul Neely, 1020 S. W. Taylor St., Portland.
Secretary-Treasurer: Dr. Lewis Jordon, 1020 S. W. Taylor St., Portland.
Place: Good Samaritan Hospital, Portland. Time: Third Tuesday of each month.

Pennsylvania Academy of Ophthalmology and Otolaryngology

President: Dr. Lewis T. Buckman, 83 S. Franklin St., Wilkes-Barre.
Secretary Pro Tem: Dr. Paul C. Craig, 232 N. 5th St., Reading.
Time: Last week in April.

Rhode Island Ophthalmological and Otological Society

Acting President: Dr. N. Darrell Harvey, 112 Waterman St., Providence.
Secretary-Treasurer: Dr. Linley C. Happ, 124 Waterman St., Providence.
Place: Rhode Island Medical Society Library, Providence. Time: 8:30 p. m., second Thursday in October, December, February and April.

South Carolina Society of Ophthalmology and Otolaryngology

President: Dr. J. L. Sanders, 222 N. Main St., Greenville.
Secretary: Dr. J. H. Stokes, 125 W. Cheves St., Florence.

Tennessee Academy of Ophthalmology and Otolaryngology

President: Dr. Wesley Wilkerson, 700 Church St., Nashville.
Secretary-Treasurer: Dr. W. D. Stinson, 124 Physicians and Surgeons Bldg., Memphis.

Texas Ophthalmological and Oto-Laryngological Society

President: Dr. F. H. Rosebrough, 603 Navarro St., San Antonio.
Secretary: Dr. M. K. McCullough, 1717 Pacific Ave., Dallas.

Utah Ophthalmological Society

President: Dr. R. B. Maw, 699 E. South Temple, Salt Lake City.
Secretary-Treasurer: Dr. Charles Ruggeri Jr., 1120 Boston Bldg., Salt Lake City.
Place: University Club, Salt Lake City. Time: 7:00 p. m., third Monday of each month.

Virginia Society of Oto-Laryngology and Ophthalmology

President: Dr. Mortimer H. Williams, 30½ Franklin Rd. S. W., Roanoke.
Secretary-Treasurer: Dr. Meade Edmunds, 34 Franklin St., Petersburg.

West Virginia State Medical Association, Eye, Ear, Nose and Throat Section

President: Dr. George Traugh, 309 Cleveland Ave., Fairmont.
Secretary: Dr. Welch England, 621½ Market St., Parkersburg.

LOCAL

Akron Academy of Ophthalmology and Otolaryngology

President: Dr. E. L. Mather, 39 S. Main St., Akron, Ohio.
Secretary-Treasurer: Dr. V. C. Malloy, 2d National Bank Bldg., Akron, Ohio.
Time: First Monday in January, March, May and November.

Atlanta Eye, Ear, Nose and Throat Society

President: Dr. B. M. Cline, 153 Peachtree St. N. E., Atlanta, Ga.
Acting Secretary: Dr. A. V. Hallum, 478 Peachtree St. N. E., Atlanta, Ga.
Place: Grady Hospital. Time: 6:00 p. m., fourth Monday of each month, from October to May.

Baltimore Medical Society, Section on Ophthalmology

Chairman: Dr. Ernst Bodenheimer, 1212 Eutaw Pl., Baltimore.
Secretary: Dr. Thomas R. O'Rourk, 104 W. Madison St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to March.

Birmingham Eye, Ear, Nose and Throat Club

President: Each member, in alphabetical order.
Secretary: Dr. Luther E. Wilson, 919 Woodward Bldg., Birmingham, Ala.
Place: Tutwiler Hotel. Time: 6:30 p. m., second Tuesday of each month, September to May, inclusive.

Brooklyn Ophthalmological Society

President: Dr. William B. Agan, 1 Nevins St., Brooklyn.
Secretary-Treasurer: Dr. Benjamin C. Rosenthal, 140 New York Ave., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. Walter F. King, 519 Delaware Ave., Buffalo.
Secretary-Treasurer: Dr. Sheldon B. Freeman, 196 Linwood Ave., Buffalo.
Time: Second Thursday of each month.

Chattanooga Society of Ophthalmology and Otolaryngology

President: Each member, in alphabetical order.
Secretary: Dr. Douglas Chamberlain, Chattanooga Bank Bldg., Chattanooga, Tenn.
Place: Mountain City Club. Time: Second Thursday of each month from September to May.

Chicago Ophthalmological Society

President: Dr. Vernon M. Leech, 55 E. Washington St., Chicago.
Secretary: Dr. W. A. Mann, 30 N. Michigan Ave., Chicago.
Place: Chicago Towers Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

Cincinnati General Hospital Ophthalmology Staff

Chairman: Dr. D. T. Vail, 441 Vine St., Cincinnati.
Secretary: Dr. A. A. Levin, 441 Vine St., Cincinnati.
Place: Cincinnati General Hospital. Time: 7:45 p. m., third Friday of each month except June, July and August.

Cleveland Ophthalmological Club

Chairman: Dr. Shandor Monson, 1621 Euclid Ave., Cleveland.
Secretary: Dr. Carl Ellenberger, 14805 Detroit Ave., Cleveland.
Time: Second Tuesday in October, December, February and April.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. W. S. Reese, 1901 Walnut St., Philadelphia.
Clerk: Dr. George F. J. Kelly, 37 S. 20th St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Dr. H. D. Emswiler, 370 E. Town St., Columbus, Ohio.
Secretary-Treasurer: Dr. D. G. Sanor, 206 E. State St., Columbus, Ohio.
Place: The Neil House. Time: 6 p. m., first Monday of each month.

Corpus Christi Eye, Ear, Nose and Throat Society

Chairman: Dr. Arthur Padillo, 414 Medical Professional Bldg., Corpus Christi, Texas.
Secretary: Dr. Edgar G. Mathis, 815 Medical Arts Bldg., Corpus Christi, Texas.
Time: Second Friday of each month from October to May.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. Ruby K. Daniel, Medical Arts Bldg., Dallas 1, Texas.
Secretary: Dr. Tom Barr, Medical Arts Bldg., Dallas 1, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. H. C. Schmitz, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Dr. Byron M. Merkel, 604 Locust St., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month from September to May.

Detroit Ophthalmological Club

Chairman: Dr. Raymond S. Goux, 545 David Whitney Bldg., Detroit 26.
Secretary: Dr. Arthur Hale, 1609 Eaton Tower, Detroit.
Place: Club rooms of Wayne County Medical Society. Time: 6:30 p. m., third Thursday of each month, November through April.

Detroit Ophthalmological Society

President: Dr. Parker Heath, 1553 Woodward Ave., Detroit.
Secretary: Dr. Leland F. Carter, 1553 Woodward Ave., Detroit.
Place: Club rooms of Wayne County Medical Society. Time: Third Thursday of each month from November to April, inclusive.

Eastern New York Eye, Ear, Nose and Throat Association

President: Appointed at each meeting.
Secretary-Treasurer: Dr. Joseph L. Holohan, 330 State St., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. Rex Howard, 602 W. 10th St., Fort Worth, Texas.
Secretary-Treasurer: Dr. R. H. Gough, Medical Arts Bldg., Fort Worth, Texas.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

Houston Academy of Medicine, Ophthalmological and Oto-Laryngological Section

President: Dr. Lyle J. Logue, 1304 Walker Ave., Houston, Texas.

Secretary: Dr. John T. Stough, 803 Medical Arts Bldg., Houston, Texas.

Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

Indianapolis Ophthalmological and Oto-Laryngological Society

President: Dr. Myron Harding, 23 E. Ohio St., Indianapolis.

Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.

Place: Indianapolis Athletic Club. Time: 6:30 p. m., second Thursday of each month from November to May.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. Edgar Johnson, 906 Grand Ave., Kansas City, Mo.

Secretary: Dr. W. E. Keith, 1103 Grand Ave., Kansas City, Mo.

Time: Third Thursday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

Chairman: Dr. Harold Snow, 614 S. Pacific Ave., San Pedro, Calif.

Secretary-Treasurer: Dr. Oliver R. Nees, 508 Times Bldg., Long Beach, Calif.

Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

Los Angeles Society of Ophthalmology and Otolaryngology

President: Dr. M. E. Trainor, 523 W. 6th St., Los Angeles.

Secretary-Treasurer: Dr. Orrie E. Ghrist, 210 N. Central Ave., Glendale, Calif.

Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:00 p. m., fourth Monday of each month from September to May, inclusive.

Louisville Eye and Ear Society

President: Dr. Joseph S. Heitger, Heyburn Bldg., Louisville, Ky.

Secretary-Treasurer: Dr. J. W. Fish, 321 W. Broadway, Louisville, Ky.

Place: Brown Hotel. Time. 6:30 p. m., second Thursday of each month from September to May, inclusive.

Lower Anthracite Eye, Ear, Nose and Throat Society

Chairman: Each member in alphabetical order.

Secretary: Dr. James J. Monohan, 31 S. Jardin St., Shenandoah, Pa.

Medical Society of the District of Columbia, Section of Ophthalmology and Otolaryngology

Chairman: Dr. P. S. Constantinople, 1835 I St. N. W., Washington.

Secretary: Dr. Frazier Williams, 1801 I St. N. W., Washington.

Place: 1718 M St. N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

Memphis Society of Ophthalmology and Otolaryngology

Chairman: Each member in alphabetical order.

Secretary: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis, Tenn.

Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month from September to May.

Milwaukee Oto-Ophthalmic Society

President: Dr. Edwin C. Bach, 324 E. Wisconsin Ave., Milwaukee.

Secretary-Treasurer: Dr. Ralph T. Rank, 238 W. Wisconsin Ave., Milwaukee.

Place: University Club. Time: 6:30 p. m., second Tuesday of each month.

Montgomery County Medical Society

Chairman: Dr. H. V. Dutrow, 1040 Fidelity Medical Bldg., Dayton, Ohio.

Secretary-Treasurer: Dr. Maitland D. Place, 981 Reibold Bldg., Dayton, Ohio.

Place: Van Cleve Hotel. Time: 6:30 p. m., first Tuesday of each month from October to June, inclusive.

Montreal Ophthalmological Society

President: Dr. J. Rosenbaum, 1396 Ste. Catherine St. W., Montreal, Canada.

Secretary: Dr. L. Tessier, 1230 St. Joseph Blvd. E., Montreal, Canada.

Time: Second Thursday of October, December, February and April.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. M. M. Cullom, 700 Church St., Nashville, Tenn.

Secretary: Dr. R. E. Sullivan, 432 Doctors Bldg., Nashvile, Tenn.

Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to May

New Haven Ophthalmological Society

President: Dr. William H. Ryder, 185 Church St., New Haven, Conn.

Secretary: Dr. Frederick A. Wiess, 255 Bradley St., New Haven, Conn.

New Orleans Ophthalmological and Oto-Laryngological Society

President: Dr. W. B. Clark, 1012 American Bank Bldg., New Orleans.

Secretary: Dr. Mercer G. Lynch, 1018 Maison Blanche Bldg., New Orleans.

Place: Louisiana State University Medical Bldg. Time: 8 p. m., second Tuesday of each month from October to May.

New York Academy of Medicine, Section of Ophthalmology

Chairman: Dr. Frank C. Keil, 660 Madison Ave., New York.
Secretary: Dr. Willis S. Knighton, 121 E. 61st St., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

New York Society for Clinical Ophthalmology

President: Dr. Milton Berliner, 57 W. 57th St., New York.
Secretary: Dr. Benjamin Esterman, 983 Park Ave., New York.
Place: Squibb Hall, 745–5th Ave. Time: 8 p. m., first Monday of each month from October to May, inclusive.

Oklahoma City Academy of Ophthalmology and Otolaryngology

President: Dr. James P. Luton, 117 N. Broadway, Oklahoma City.
Secretary: Dr. Harvey O. Randel, 117 N. Broadway, Oklahoma City.
Place: University Hospital. Time: Second Tuesday of each month from September to May.

Omaha and Council Bluffs Ophthalmological and Oto-Laryngological Society

President: Dr. D. D. Stonecypher, Nebraska City, Neb.
Secretary-Treasurer: Dr. W. Howard Morrison, 1500 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m. dinner; 7 p. m. program; third Wednesday of each month from October to May.

Passaic-Bergen Ophthalmological Club

President: Dr. Thomas Sanfacon, 340 Park Ave., Paterson, N. J.
Secretary-Treasurer: Dr. J. Averbach, 435 Clinton Ave., Clinton, N. J.
Place: Paterson Eye and Ear Infirmary. Time: 9 p. m., last Friday of every month, except June, July and August.

Philadelphia County Medical Society, Eye Section

President: Dr. Isaac Tassman, 136 S. 16th St., Philadelphia.
Secretary: Dr. Glen Gregory Gibson, 255 S. 17th St., Philadelphia.
Time: First Thursday of each month from October to May.

Pittsburgh Ophthalmological Society

President: Dr. George H. Shuman, 351-5th Ave., Pittsburgh.
Secretary: Dr. Robert J. Billings, 509 Liberty Ave., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

Reading Eye, Ear, Nose and Throat Society

President: Dr. R. M. Brickbauer, Shillington, Pa.
Secretary: Dr. Paul C. Craig, 232 N. 5th St., Reading, Pa.
Place: Wyomissing Club. Time: 6:30 p. m., third Wednesday of each month from October to July.

Richmond Ophthalmological and Oto-Laryngological Society

President: Dr. Peter N. Pastore, Medical College of Virginia, Richmond, Va.
Secretary: Dr. Clifford A. Folkes, Professional Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

Rochester Eye, Ear, Nose and Throat Society

President: Dr. Frank Barber, 75 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. Charles T. Sullivan, 277 Alexander St., Rochester, N. Y.

St. Louis Ophthalmic Society

President: Dr. C. C. Beisbarth, 3720 Washington Blvd., St. Louis.
Secretary: Dr. H. R. Hildreth, 508 N. Grand Blvd., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting, 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

San Antonio Ophthalmo-Oto-Laryngological Society

President: Dr. Belvin Pritchett, 705 E. Houston St., San Antonio 5, Texas.
Secretary-Treasurer: Lt. Col. John L. Matthews, AAF School of Aviation Medicine, Randolph Field, Texas.
Place: San Antonio, Brooke General Hospital, Randolph Field or San Antonio Aviation Cadet Center. Time: 7 p. m., second Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Roy H. Parkinson, 870 Market St., San Francisco.
Secretary: Dr. A. G. Rawlins, 384 Post St., San Francisco.
Place: Society's Bldg., 2180 Washington St., San Francisco. Time: Fourth Tuesday of every month except June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. David C. Swearingen, Slattery Bldg., Shreveport, La.
Secretary-Treasurer: Dr. Kenneth Jones, Medical Arts Bldg., Shreveport, La.
Place: Shreveport Charity Hospital. Time: 7:30 p. m., first Monday of every month except July, August and September.

Spokane Academy of Ophthalmology and Oto-Laryngology

President: Dr. Clarence A. Veasey Sr., 421 W. Riverside Ave., Spokane, Wash.
Secretary: Dr. Clarence A. Veasey Jr., 421 W. Riverside Ave., Spokane, Wash.
Place: Spokane Medical Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. A. H. Rubenstein, 713 E. Genesee St., Syracuse, N. Y.
Secretary-Treasurer: Dr. I. H. Blaisdell, 713 E. Genesee St., Syracuse, N. Y.
Place: University Club. Time: First Tuesday of each month except June, July and August.

Toledo Eye, Ear, Nose and Throat Society

Chairman: Dr. E. W. Campbell, 316 Michigan St., Toledo, Ohio.
Secretary: Dr. L. C. Ravin, 316 Michigan St., Toledo, Ohio.
Place: Toledo Club. Time: Each month except June, July and August.

Toronto Academy of Medicine, Section of Ophthalmology

Chairman: Dr. W. R. F. Luke, 316 Medical Arts Bldg., Toronto, Canada.
Secretary: Dr. W. T. Gratton, 216 Medical Arts Bldg., Toronto, Canada.
Place: Academy of Medicine, 13 Queens Park. Time: First Monday of each month, November to April.

Washington, D. C., Ophthalmological Society

President: Dr. S. Bockoven, 1752 Massachusetts Ave., Washington, D. C.
Secretary-Treasurer: Dr. John Lloyd, 1218–16th St. N. W., Washington, D. C.
Place: Medical Society of District of Columbia Bldg., 1718 M St. N. W., Washington, D. C. Time: 7:30 p. m., first Monday in November, January, March and May.

Wilkes-Barre Ophthalmological Society

Chairman: Each member in turn.
Secretary: Dr. Samuel T. Buckman, 70 S. Franklin St., Wilkes-Barre, Pa.
Place: Office of chairman. Time: Last Tuesday of each month from October to May.